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AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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DEPARTMENTS

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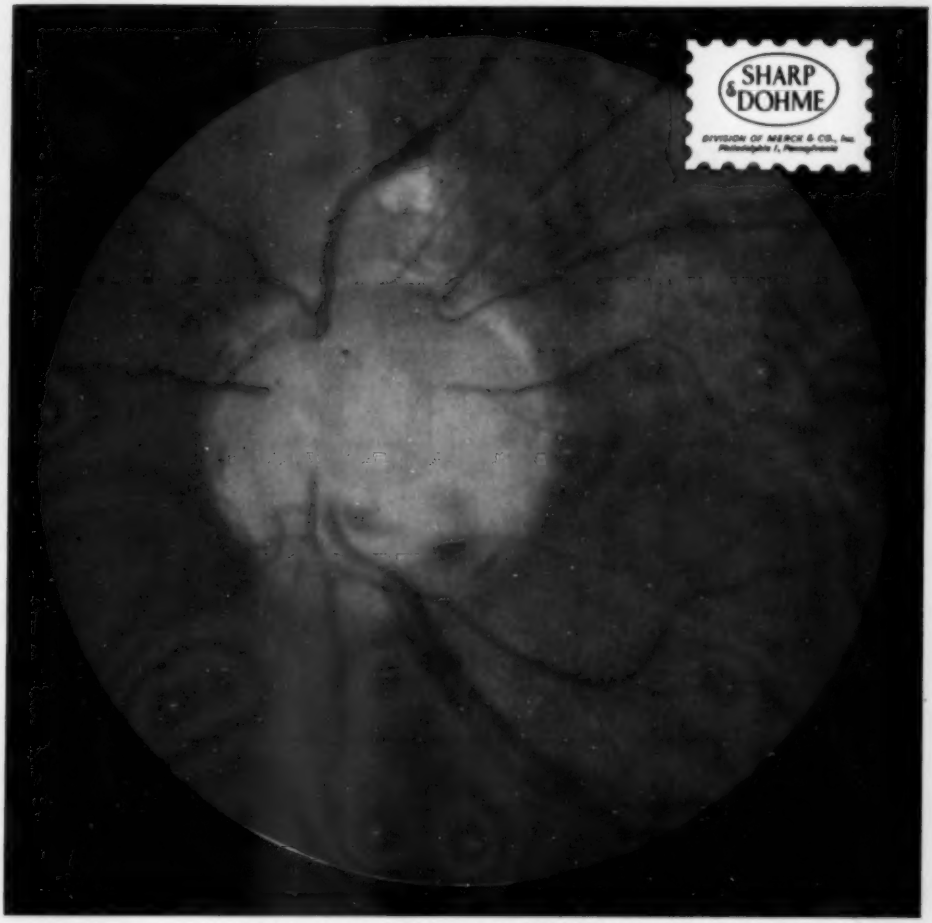

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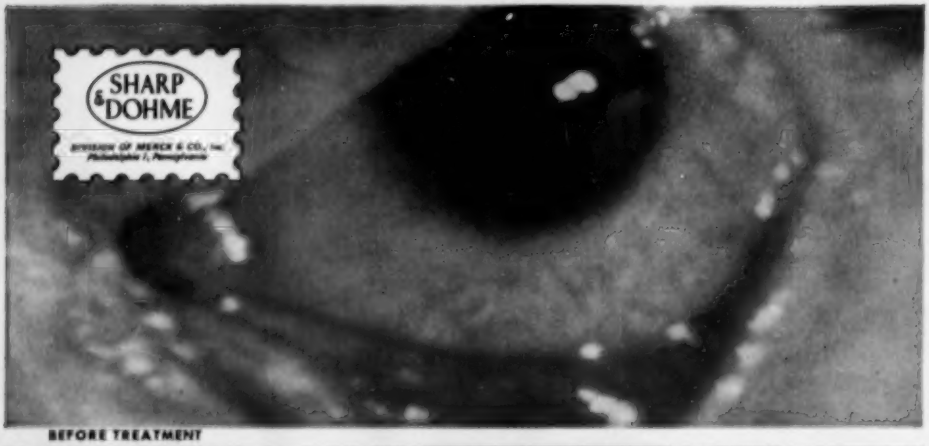

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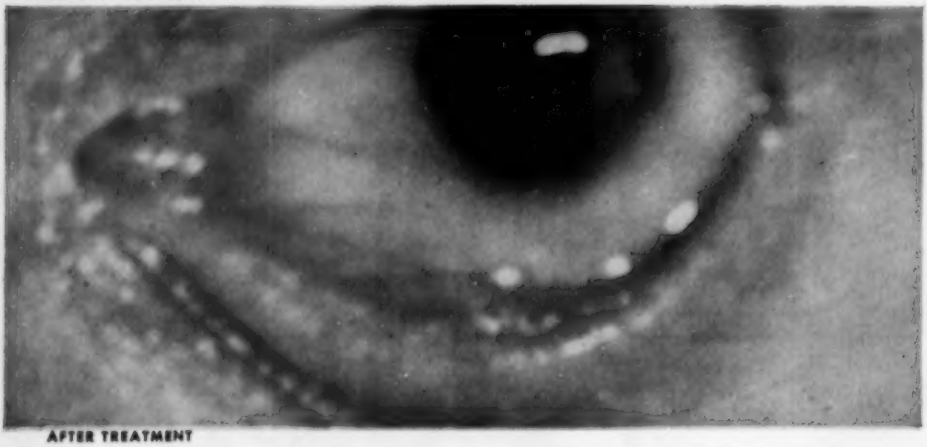

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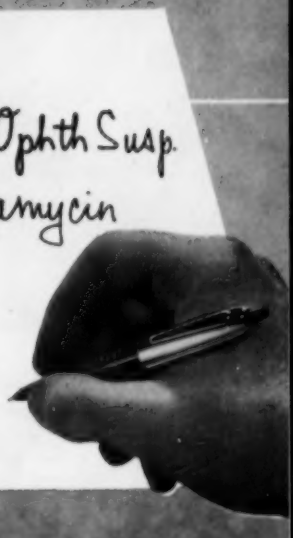

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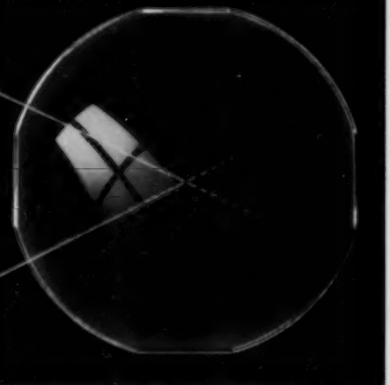

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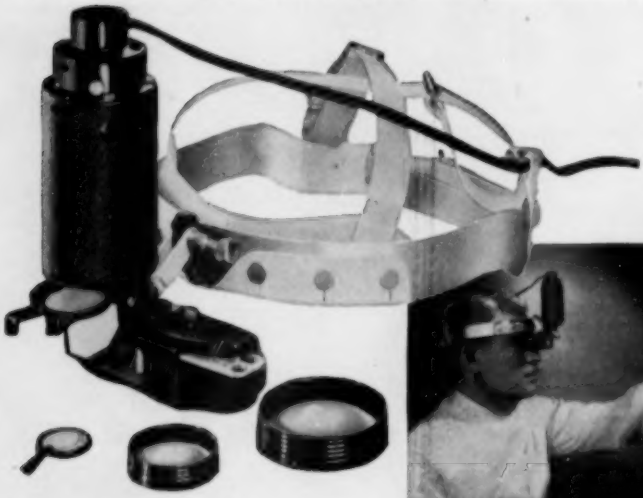

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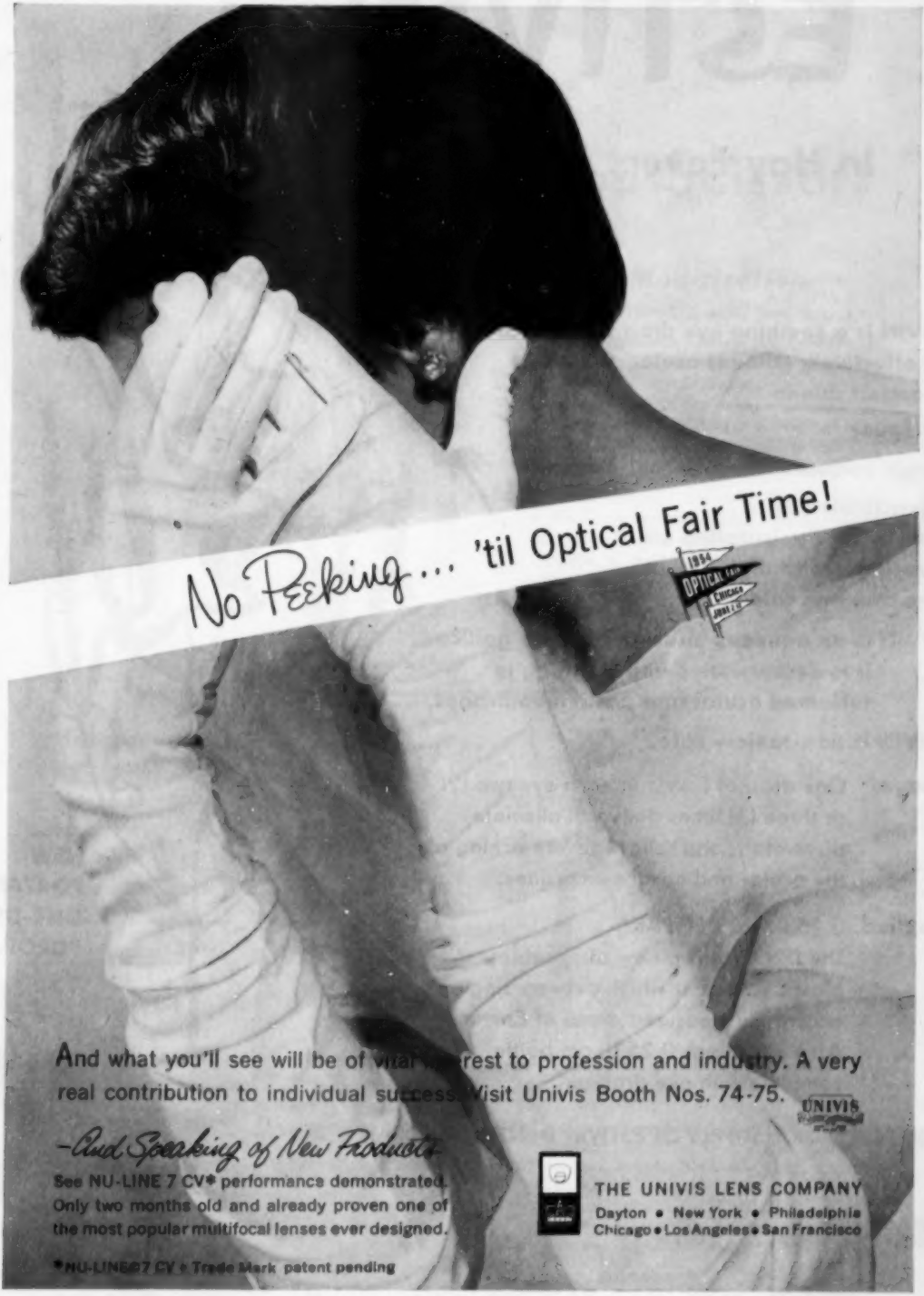
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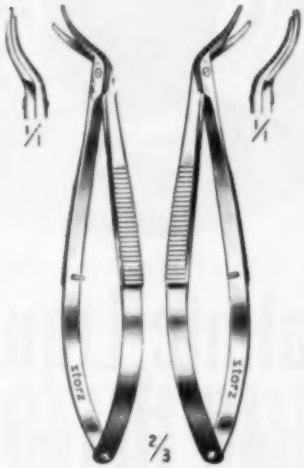

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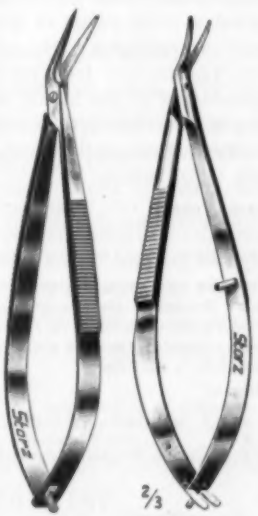

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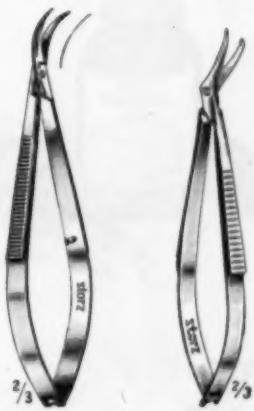

475 481

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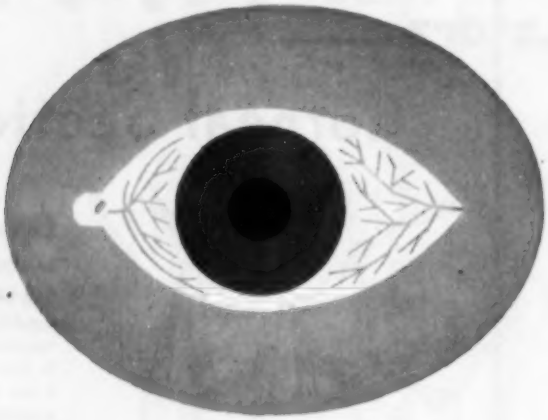
ophthalmic
infections...



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Furacin Ophthalmic Ointment contains Furacin 1% in a petrolatum type base. ¼ oz. tube.

References

1. Pritikin, R. I.: J. Internat. Coll. Surgeons 17: 234, 1952.
2. _____, and Duchon, M. L.: Mil. Surgeon 109: 706, 1951.
3. _____, _____, and Farmer, H. S.: Mil. Surgeon 108: 309, 1951.
4. Brennan, J. W.: Am. J. Ophth. 35: 1343, 1952.

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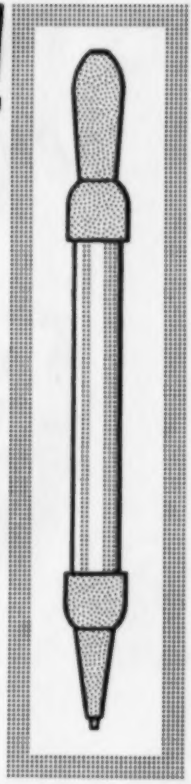

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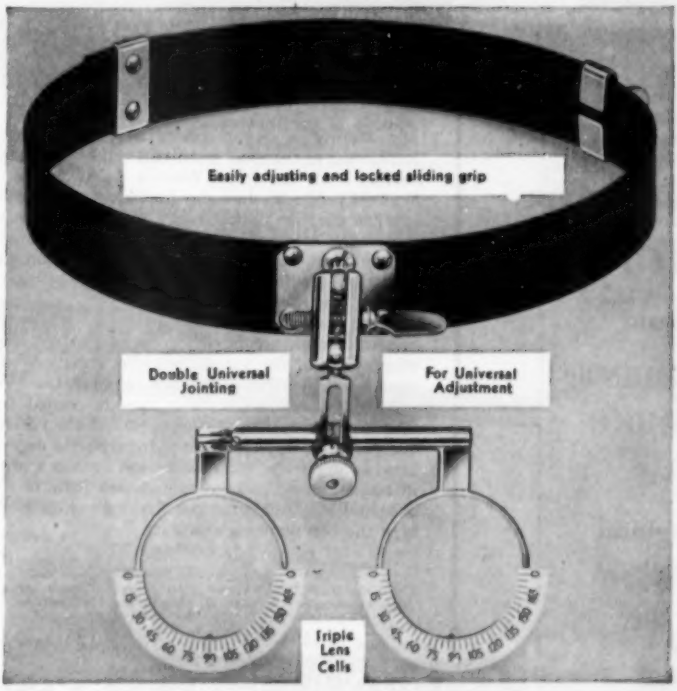

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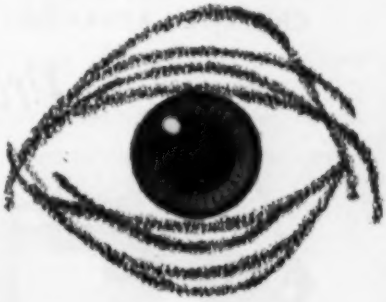

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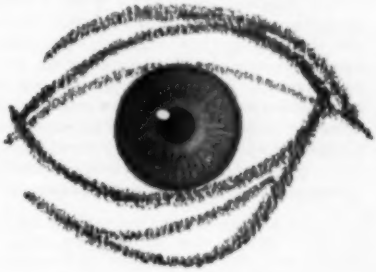

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1. Gettes, B. C., and Leopold, I. H.: A. M. A. Arch. of Ophth. 49:24 (Jan.) 1953.
2. Stolzar, I. H.: Am. J. Ophth. 36:110 (Jan.) 1953.

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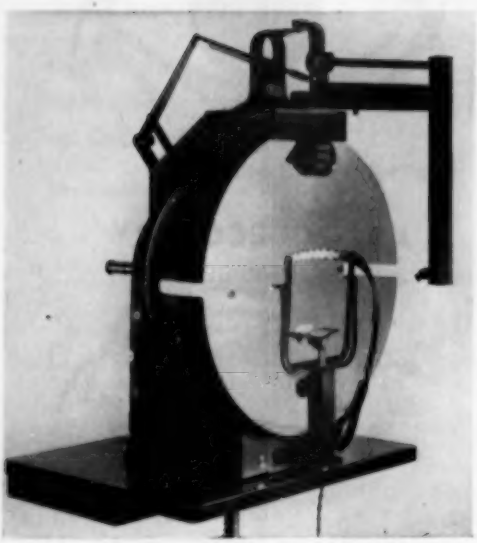

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Uniform illumination and variable at will. Complete independence of illumination of perimeter bowl from light outside as well as controlled constancy of contrast between basic illumination and brightness of object are some of the advantages. Targets of various sizes and colors and always exactly the same reproducible contrast are projected on field of perimeter. Movement of the targets is controlled by means of a handle which can be moved in any direction over a perimeter record chart. Position of handle on chart corresponds to position of target in hemisphere.

Almark Projection Perimeter —Curry & Paxton

The test stimulus is of uniform brightness and is made to travel centrally along the arc up to 100° on either side of central fixation object. Sixty-four different test stimuli may be presented by combinations of three control wheels on top of lamp housing. Apertures in wheel are all clear but of different sizes. Apertures in second wheel change colors from white to red, green or blue. Third wheel contains one free aperture and 3 free neutral filters of different optical densities. By means of a silently operated spring-loaded lever the stimulus in any part of an arc may instantaneously be extinguished or revealed by a flicker device. Recording is automatic.

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CONTENTS

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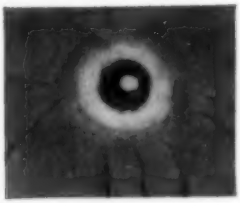

FIG. A

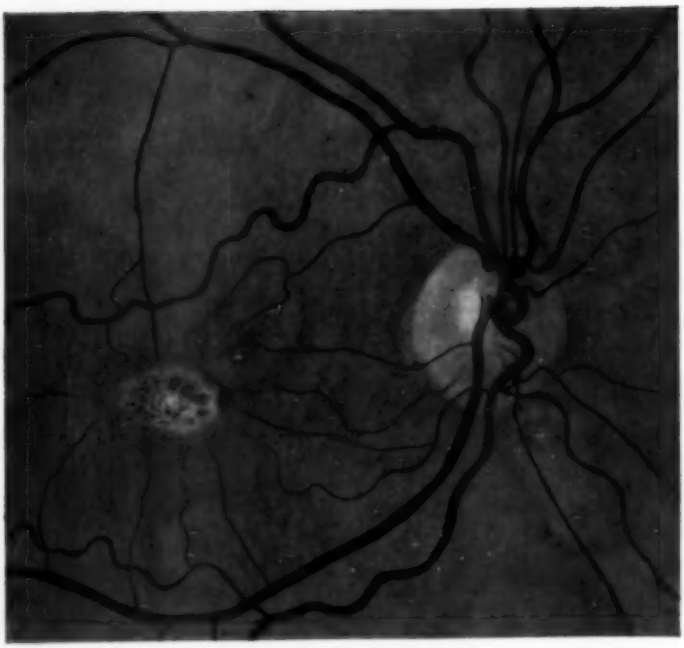

FIG. B

FIGS. A AND B (DELLAPORTA). (FIG. A) ENDODIATHERMY COAGULATION SEALING THE MACULAR HOLE. THE DRAWING WAS MADE IMMEDIATELY AFTER THE TRANSBULBAR NEEDLE WAS WITHDRAWN. (FIG. B) COAGULATION SCAR THREE MONTHS LATER.

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ENDODIATHERMY*

A METHOD FOR SEALING MACULAR HOLES BY TRANSBULBAR COAGULATION

ANGELOS DELLAPORTA, M.D.
Buffalo, New York

Most ophthalmologists will agree that the surgical treatment of macular holes is very difficult and not always successful. The main reasons are the difficulty of exact localization and the unsatisfactory exposure of the posterior pole of the globe.

Peripheral retinal tears can be accurately localized by the transillumination method of Weve-Lindner,[1] but this method is not easily applicable to macular holes because the space conditions of the surgically exposed posterior pole of the eyeball are very limited. Even in cases in which the assisting surgeon is able to spot the light beam of the ophthalmoscope on the surface of the sclera, the small size of the macular hole compared to the light beam does not permit accurate localization. Usually only the approximate position of the macula on the bared sclera is determined.

For this purpose, Lindner[1] measures 2.5 mm. laterally from the optic nerve toward the insertion of the inferior oblique and Böck[2] measures 2.0 mm. nasally from the medial end of the insertion of the inferior oblique; Pillat uses the instrument devised by Carsten[3] which consists of a thin wire, curved to fit the curvature of the sclera and equal in length to the surface distance between limbus and macula.

After the macula is approximately determined by one of these methods, a so-called orientating coagulation is applied at the corresponding spot of the sclera either by diathermy or catholysis current, the latter being preferred because it produces a smaller scar.

The position of the orientating coagulation in relation to the macular hole is checked by the ophthalmoscope and the subsequent punctures are calculated to hit the macular hole. These coagulations should be made by diathermic current because this produces firmer adhesions. The whole procedure is difficult because the posterior pole of the eye is not easily accessible and the coagulations are not easily recognized with the ophthalmoscope.

Strampelli[4] directs the light of a specially designed lamp of Lange's type over the sclera of the macular region while the surgeon observes the macular hole with the ophthalmoscope. The lamp produces a particularly small focused light; its tip, being constructed of noninsulated metal, can be used as a dull diathermic electrode.

The undermining procedure (Unterminierungsmethode), invented by Lindner,[1,5] is another method for sealing macular holes. The chorioretinal adhesions are produced by chemical action of three-percent potassium-hydroxide solution injected between sclera and choroid at the area of the macular hole. The localization is made by transillumination but, for this procedure, an approximate localization is sufficient. The undermining method is claimed to offer the best final vision results; it also is a difficult operation and can be very dangerous unless exactly performed.

Because all these procedures are difficult, require great skill, and often are not accurate, they have not become popular. In

* From the Eye-Bank and Research Laboratory, University of Buffalo Medical School. Dedicated to Prof. Karl Lindner on the occasion of his 70th birthday.

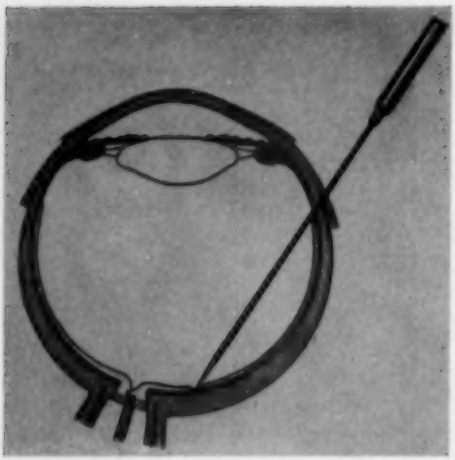

Fig. 1 (Dellaporta). Through a small opening at the ora serrata, the needle is introduced into the globe, directed at the desired spot of the eyeground under ophthalmoscopic observation, and its point pricked into the globe's wall.

dealing with retinal detachments with macular holes, the ophthalmologist was and still is confronted with a formidable situation.

In a series of experiments[6] on rabbits' eyes, I tried to determine if it is possible to localize on the sclera a given point of the posterior half of the fundus with a transbulbar needle under ophthalmoscopic observation. The conclusions were:

1. Any spot in the posterior half of the fundus seen with the ophthalmoscope can be exactly localized on the outer surface of the sclera with the help of a long transbulbar needle. The needle is 30-mm. long and is entirely insulated by lacquer, except for its three-mm. long point.

Through a small opening at the ora serrata, the needle is introduced into the globe, directed at the desired spot of the eyeground under ophthalmoscopic observation, and its point pricked into the globe's wall (fig. 1). The point of the needle which perforates the previously exposed sclera indicates the corresponding spot of the fundus.

The perforation point is marked with gentian violet and, after withdrawing the needle, diathermy is applied there; in cases of flat detached retina, this will invariably hit the hole. In a similar way a foreign body or any other structure on the retina can be localized with the transbulbar needle.

2. In retinal holes, coagulation can be produced directly by transmitting diathermic current to the sticking point of the transbulbar needle (endodiathermy). In this case, care must be taken that the lacquer limit lies in front of the choroid so that the naked point of the needle contacts the sclera and choroid.

3. The intensity and time of action for the diathermic current to produce such transbulbar coagulations are the same as those required for diascleral diathermic puncture.

4. The actual production of the coagulations can be observed with the ophthalmoscope. Their size depends on the time the diathermic current is allowed to act. Two to eight seconds produces coagulated areas of one-third to four disc diameters.

5. Transbulbar coagulation does no more damage than diascleral coagulation. In no case was the vitreous body injured by this method.

These experiments led to the development of the following clinical procedure which I have successfully performed in three cases.

Surgical procedure

Preliminary remarks

The transbulbar needle* is 30-mm. long and 0.5-mm. thick. It is insulated with lacquer, except for its point which is three-mm. long and 0.2 to 0.3-mm. thick. The needle is mounted on a naked, round shaft, 12-mm. long and 2.0-mm. thick. Electric current can be transmitted only through the shaft to the naked point of the needle.

Special care must be taken that the insulating lacquer is not defective. To test this, the needle is fixed on an ordinary electrode handle, put into distilled water, and ca-

* Manufactured by C. Reiner, Mariannengasse 17, Vienna 9.

tholysis current sent through it. Hydrogen bubbles will immediately appear at any defective spot on the insulated part of the needle and, of course, at the naked point of the needle. The needle is made of rigid steel and is sterilized by dry heat.

Connection with the electric current is established by a very thin, 20-cm. long, naked copper wire whose ends must be polished for easy conduction of the electric current. One end of the copper wire is passed through a 12-mm., thin rubber tube which is subsequently drawn over the needle's shaft. The rubber tube presses the copper wire firmly against the shaft of the needle and also insulates the latter (fig. 2). The other end of the copper wire is attached by a hemostat to the operating field drape. Before the operation starts, the lead plate, the passive pole of the diathermic apparatus, is fixed to the flank or leg of the patient.

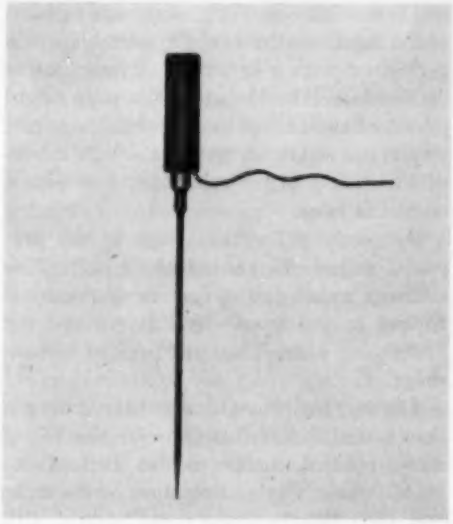

Fig. 2 (Dellaporta). The transbulbar needle with rubber tube on the shaft and connecting copper wire.

Preoperative care

Rest in bed with stenopeic spectacles is recommended in order to flatten the detached retina. Repeated fundus examinations for detection of peripheral tears is important, as many of the macular holes are of secondary origin.

Preparation for operation

Maximal pupil dilatation is achieved by repeated instillations of atropine and, if necessary, of 10-percent neosynephrine. The patient lies flat on a low operating table, his head resting on a small pillow in such a position that the surgeon, standing behind the patient's head, can easily carry on direct ophthalmoscopy.

Anesthesia

A three-percent cocaine solution is instilled once before and once after the washing of the eye. Cocaine instillations are restricted in order to protect the transparency of the cornea. Two-percent novocaine with adrenalin is used to infiltrate the external lid angle and the retrobulbar space, and 0.3 cc. is also injected subconjunctivally over the insertion of each rectus muscle.

Operation

After canthotomy of the external lid angle, a small lid speculum is inserted. The sclera is exposed about the lateral rectus as in tenotomy and the muscle is freed up to five mm. from its insertion. A traction suture is passed through this muscle and another one through the intact conjunctiva and underlying inferior rectus. These ligatures and the anesthesia with two-percent novocaine provide complete immobility of the eyeball, which is necessary for the operation.

The sterilized transbulbar needle, armed with the rubber tube and the copper wire, as already described, is then prepared. The free end of the copper wire is grasped with a hemostat and fastened to the dry operating field drape.

The perforation of the eyeball at the ora serrata follows. The insertion of the lateral rectus is partially resected on the lower end,

and here a four-mm. long meridional incision of the outer two thirds of the scleral layers is performed with a keratome. A long Suarez de Mendoza (like McLean), 4-0, plain catgut preplaced suture is passed through the scleral wound lips and the catgut thread is pulled out of the groove of the scleral incision with a blunt iris hook.

By gently pulling the loops of the preplaced suture, the ground of the scleral incision is exposed, and surface diathermy is applied to the deep scleral layers and the underlying uvea. This will prevent hemorrhage.

The surgeon places himself behind the patient's head and adjusts the refraction of his direct ophthalmoscope to the level of the macular hole. For an operation on the right eye, the ophthalmoscope must be held with the left hand, for an operation on the left eye, with the right hand. The ophthalmoscope is then put aside.

The proper transmission of current through hemostat, copper-wire, transbulbar needle is checked by cauterizing the conjunctiva with the point of the needle. In order to close the current, the assistant touches the hemostat, which is fastened to the copper wire, with the ball-shaped electrode.

In the coagulated groove of the scleral incision, a small opening is then made with a keratome. The opening should be large enough for the transbulbar needle to pass through without any resistance.

The surgeon holds the ophthalmoscope again with the appropriate hand, takes the shaft of the transbulbar needle between thumb and index finger of the other hand, and introduces the needle into the eyeball. He directs the needle's point toward the center of the eye in order to avoid damage to the lens or the eye membranes. *During this step he watches the pupillary area with the naked eye under oblique illumination with the light beam of the ophthalmoscope, with the operating room darkened.*

When the needle is introduced about 10 mm. into the eye, its shining point, magnified through the refractive power of the eye, appears behind the lens. At this moment, the oblique illumination is discontinued.

The surgeon puts the ophthalmoscope to his eye and begins ophthalmoscopic observation of the needle. The refraction of the ophthalmoscope should not be changed because the needle is sufficiently visible with this adjustment. *The surgeon watches the macular hole and pushes the needle slowly forward until its point is seen some millimeters above the macula.*

This operative step is facilitated by observing the following points:

1. *The refraction of the ophthalmoscope.* The surgeon watches the macular hole with the ophthalmoscope (adjusted at the level of the macular hole) and at first sees the needle shadowlike but, the closer the needle point approaches the retina, the sharper it appears. When the point of the needle appears as sharp as the macular hole, then the point is very close to the retina because it has reached the refractive level of the retina.

2. *The shadow of the needle on the fundus.* The point of the transbulbar needle illuminated with the ophthalmoscope's beam casts a shadow on the fundus. This shadow is the most dependable leader for directing the needle to the macular hole. *The surgeon watches the needle's point and its shadow coming closer; when they touch each other, the point of the needle has actually reached the retina.*

3. After some practice, the direction in which the needle has to be pushed forward is sensed with the fingers.

The pricking of the needle into the eye wall. When the needle point is close to the macular hole, the only allowed movements are the forward-pushing or backward-pulling of the needle in the direction which is determined by the straight line, connecting the scleral perforation at the ora serrata and the macular hole. Movement of the shaft of the needle in any sideward direction is dangerous

as the inner membranes of the eye may be injured by its point.

The point is then passed through the macular hole, to the choroid, and pushed slowly but firmly forward until the sclera is perforated. The actual perforation of the sclera can be felt by the fingers. The lacquer limit must be just in front of the choroid.

Now the ophthalmoscopic observation can be interrupted and the shaft of the needle released. *Between the introduction of the needle into the eyeball and the pricking of its point into the sclera, the shaft of the needle must be held permanently by the surgeon's fingers.* If the needle is released earlier, the heavier shaft will tilt and the needle point may injure the lens or the inner membranes of the eye.

The actual production of the coagulation. The surgeon watches the macular hole with the ophthalmoscope and asks the assistant to close the current. Small air bubbles immediately appear at the lacquer limit of the needle and, simultaneously, a rapidly growing coagulation of the choroid and the retina is produced.

As soon as the borders of the retinal hole start to show the well-known whitish-yellow color, the current is shut off. For typical macular holes, two to three seconds of current action are enough to produce an appropriate coagulated area of one-third to one-half disc diameters. If the current is allowed to act longer, the coagulation increases rapidly in size, causing undesirable damage of the macular retina.

The adjustment of the diathermic apparatus producing the current should be the same as is usually administered for producing ophthalmoscopically visible diascleral retinal coagulations. In typical macular holes, the retina lies close to the choroid so that both are simultaneously coagulated. When the point of the needle has been passed through the center of the macular hole, one can see the shining point surrounded by a small red ring, which is the choroid seen through the retinal hole and around it the whitish-yellow ring of the coagulated retina (color plate, fig. a).

The withdrawal of the needle must be carried out slowly, under ophthalmoscopic observation. The shaft of the needle is grasped with thumb and index finger and carefully withdrawn in the same axis as its introduction. The perforation is ophthalmoscopically visible as a round white spot lined by a fine, incomplete grayish ring. Its size is about one-third to one-half of the size of a typical macular hole (color plate, fig. a).

Completion of the operation. The preplaced catgut suture in the scleral wound lips is tied and the conjunctiva and the canthotomy wounds are sutured. Penicillin may be given after surgery, atropine ointment is instilled, and both eyes are bandaged. The operation takes 15 to 20 minutes.

Postoperative treatment: Dressing the eyes is done every second day. Both eyes are kept closed for 14 days. During this time, the patient must lie flat in bed. The postoperative irritation of the eye is very mild. The remaining postoperative care is the same as in ordinary retinal detachment operation.

Report of cases

Case 1

A 61-year-old woman (M. K., No. 886/51) was admitted on August 23, 1951, with retinal detachment covering the temporal half of fundus. At the periphery of the retina at the 8-o'clock position, a round hole of 0.5 disc diameter, and a typical macular hole were found. Vision: +2.0D. sph. = 6/36, J5. The nasal half of the visual field was absent. The left eye was normal.

Operation on August 25, 1951. Canthotomy, exposing the temporal half of the globe, and temporary resection of the external rectus were done. The peripheral hole was localized with the transillumination method of Weve-Lindner and sealed by five diascleral nonperforating diathermy applications. The macular hole was sealed by a

single transbulbar diathermy application according to the technique already described.

By allowing the current to act for two to three seconds, a coagulation of one-third disc diameter was produced (color plate, fig. a). After the needle was withdrawn, four perforating diathermy applications behind the peripheral hole were employed in order to drain the subretinal fluid. The postoperative course was uneventful.

The last follow-up examination on August 4, 1952, 12 months after the operation, showed the retina completely attached, with partially pigmented coagulation scars at the periphery. In the macula a whitish-brown, slightly pigmented coagulation scar of one-fourth disc diameter was visible (color plate, fig. b). Vision was: +1.0D. sph. = 6/36, J7. The visual field was normal except for a small central scotoma.

CASE 2

A 51-year-old woman (J. F., No. 1008/51) had poor vision of the right eye since childhood. At a previous examination in 1946, a keloid corneal scar was noted on the right eye. Vision was 1/60, and with a −15.0D. sph. = 6/36, J3.

Examination on her admission on October 9, 1951, showed, besides the corneal scar, a central flat retinal detachment with a typical macular hole surrounded by cystoid degenerated retina. The optic disc was surrounded by some choroidal atrophy. The vision was limited to counting fingers at 40 cm. and the visual field showed a 15- to 20-degree absolute central scotoma for a 10-mm. white object. The left eye was normal, with vision of 6/6 and with a +2.0D. sph. J1.

Operation on October 12, 1951. The macular hole was sealed by the method of endodiathermy; the diathermic current was allowed to act for three seconds. A coagulation the size of 0.5 disc diameter was produced. The postoperative course was uneventful.

Ten days after the operation the coagulated area was flat, brown-grayish, and surrounded by a small ring of atrophic choroid, which increased in the following days. One month after the operation, a brownish pigmented coagulation scar of a size of one-third disc diameter, surrounded by a ring of atrophic choroid of a width of one-third to one-half disc diameter, was visible. The retina was attached, the vision with a −10D. sph.-3/60,J13.

On August 4, 1952, 10 months after the operation, the retina was attached, the fundus picture and vision were unchanged. Visual field was normal except for an eight-degree absolute central scotoma.

The small, ring-shaped atrophy of the choroid which developed around the coagulation scar after the operation was probably caused by the pre-existing abnormal stretching of the choroid from the high myopia. This conclusion is justified by the fact that such atrophic changes around endodiathermic coagulations were not seen in normal eyes either in animals or in men.[6]

CASE 3

A lawyer (J. M., No. 1249/51), aged 69 years, had been suffering from myopia since childhood. Flickering in the right eye for two months was followed by a retinal detachment of the temporal half of the fundus with a one disc-diameter tear located four disc diameters nasally from the optic disc. The refraction of the disc was −11D. sph., that of the tear −2.0D. sph. Vision was 6/18, J2, with correction; the temporal half of the visual field was absent.

Operation on November 13, 1951. A single diathermy application was applied with the transbulbar needle, the current being allowed to act seven to eight seconds. After the coagulation, the retina showed no changes because it was not close enough to the choroid. An uneventful postoperative course was followed by complete reattachment of the retina.

Two months later there was a reddish-brown, round coagulation scar, three disc

diameters in size, with atrophy of the pigment epithelium and sclerosed choroidal vessels. In the center a small white spot indicated the perforation of the sclera. Vision was 6/8, J1, with correction. The visual field showed a small sector-shaped constriction superotemporally.

On April 1, 1952 (four and one-half months later), a macular hemorrhage had deteriorated the vision to 6/24. The last examination, made on August 7, 1952, nine months after the operation, showed an attached retina and remnants of the macular hemorrhage. Vision was 6/18, J3, with correction.

This case shows that large coagulations, up to a size of three disc diameters, can be produced with transbulbar diathermy application when the current acts for an adequate time.

Summary

Two cases of macular holes and one of nasal tear were successfully treated with endodiathermy. The last follow-up examination conducted nine to 12 months after the operation showed that, in each of the three cases, the retina was in normal position. Judging from the clinical appearance, it can reasonably be concluded that the cure would be of lasting effect.

Comment

Mamoli[7] was the first to use the principle of transbulbar coagulation in 1937, followed by Bangerter[8] in 1940. Each author treated one case, using a needle similar to that described herein, but attached to an electrode handle. Mamoli directed the needle under ophthalmoscopic observation, Bangerter under observation with the contact glass and the slitlamp of Goldmann, at the sitting patient. These procedures displayed difficulties and complications which I also encountered in my own preliminary experiments.[6] They will be summarized briefly so that they may be avoided.

1. No electrode handle should be used for attaching the transbulbar needle because it interferes with the ophthalmoscopic observation. The angle between the ophthalmoscope-beam light and the electrode handle is so narrow that it is impossible to watch the retinal hole and the point of the needle simultaneously.

The electrode handle and the connecting electric cord are too heavy for the delicate transbulbar needle. It is not possible to hold the electrode handle in exactly the same direction as the transbulbar needle when the latter is sticking into the choroid-sclera.

The slightest tilting of the electrode handle will curve the needle, and, when its point is withdrawn from the rigid sclera in the macula, it may spring up and injure the choroid and the retina. Moreover, the use of electrode handle has the disadvantage that the surgeon is compelled to carry on the various steps of the operation—the introduction of the needle, directing the needle to the macular hole, the perforation of the choroid-sclera, the actual production of the coagulation and the withdrawal of the needle—in one continuous manipulation.

2. The transbulbar needle should not be introduced into the globe through the sclera with active diathermy current, as in Mamoli's and Bangerter's procedure, because this may result in coagulation and ensuing opacification of the vitreous body. Moreover, a perforation of the sclera made with the point of the needle alone is too small for the thicker body of the needle itself. Pushing or pulling of the needle moves the whole eye simultaneously, making fine manipulations difficult.

3. The needle point should stick in the choroid-sclera during the coagulation. When the point of the needle is not pricked into the sclera, but only put on the choroid, as in Mamoli's and Bangerter's procedures, coagulation and opacification of the vitreous body may happen. Besides, it is difficult or impossible to judge the intensity of the coagulation properly and, if during the coagula-

tion the point of the needle unintentionally contacts the retina, the latter will adhere to it and will be torn when the needle is withdrawn from the globe. Böck[9] had this experience when using Bangerter's method in experimental work.

All these complications are prevented in the method described here, as no electrode handle is used, the needle is introduced through a surgically preformed opening, and the needle point is sticking into the choroid and sclera during the coagulation.

The advantages of this operation of endodiathermy compared to previous methods are:

1. The operation lasts only 15 to 20 minutes.
2. It is an accurate method.
3. The necessary skill can be obtained in every detail on animal eyes.
4. It is not necessary to expose the posterior pole of the eye. Hence there is no danger of injuring the orbital nerves and vessels.
5. The macular hole is sealed with a single diathermy application, thus sparing the precious macular region.
6. The actual production of the coagulated area can be watched with the ophthalmoscope and its size limited by the surgeon.
7. There is negligible postoperative irritation of the eye.

Summary

A discussion of various methods of treatment for macular holes has been given. A new method of endodiathermy has been described which seals retinal holes with a transbulbar needle under direct observation with the ophthalmoscope. It is a more accurate, faster, and safer method than those commonly used. Three cases of retinal holes at the posterior pole of the eye have been reported successfully treated with it.

University of Buffalo
Medical School (14).

The cases here reported were operated at the First Eye Department of the University of Vienna, Director Prof. A. Pillat, and were presented in succession to the Ophthalmological Society of Vienna at the meetings of November 12, 1951, January 10, 1952, and February 11, 1952.

I am indebted to F. Enke, publisher of the *Klinische Monatsblätter für Augenheilkunde* for the permission to use the Figures 1 and 2.

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SPONTANEOUS RUPTURE OF THE ANTERIOR HYALOID MEMBRANE FOLLOWING INTRACAPSULAR CATARACT SURGERY

RALPH E. KIRSCH, M.D., AND WILLIAM STEINMAN, M.D.
Miami, Florida

Spontaneous postoperative rupture of the anterior hyaloid membrane following uncomplicated intracapsular cataract extraction is universally considered to be innocuous. It is the chief purpose of this paper, however, to show that this complication has led to certain pathologic changes which have seriously interfered with the anatomic and functional success of the cataract operation. Moreover, this study has provided an opportunity to observe carefully the frequency with which this complication occurs, for there are quite dissimilar reports appearing in the literature.

The earliest reference to postoperative hyaloid rupture was by Elschnig[1] who not only described the herniation of the hyaloid, later elaborated upon by Reese,[2] but mentioned free flocculi of vitreous often present in the anterior chamber.

Vannas[3] found eight cases of ruptured anterior hyaloid in 100 recently operated eyes and observed 10 eyes with tears or holes in the anterior hyaloid membrane out of 32 eyes examined two months to four years following uncomplicated intracapsular operation. It was his opinion that this rupture occurred chiefly in those eyes in which the vitreous was previously diseased, and that most of these breaks occurred in the region of the pupillary margin because of the mechanical injury in this area.

McDonald[4] and Kwaskowski[5] wrote of the occasional rupture of the hyaloid membrane with vitreous extending forward to the cornea without damage.

Knapp[6] in his classical paper on intracapsular extraction discussed the "baring of the vitreous" at some length; he states that the pupillary area may contain loose vitreous, and "excessive opacification of the vitreous prolapsed into the anterior chamber through gaps in the hyaloid membrane is sometimes observed. These masses are cloudy but do not interfere with the patient's vision and in the course of years undergo but very little change."

Hughes and Owens,[7] in their careful study of postoperative complications of cataract surgery, state that rupture of the anterior hyaloid face occurred in a number of patients several months after normal healing had occurred and resulted in no residual damage. They state that it "usually occurred after round-pupil intracapsular extractions and is probably due to pinching off the bleb of vitreous which frequently projects through the pupil."

Guyton[8] mentions briefly that "over 50 percent of patients who have round-pupil intracapsular extractions develop a break in the anterior hyaloid membrane some months after operation," but ascribes no clinical importance to the rupture.

Reese[2] reports only "an occasional instance in which a spontaneous rent in the membrane was noted months after the extraction," again without damage to the eye.

Kirby[9] states that in his experience postoperative hyaloid rupture is rare, while Arruga[10] observes that it is a complication generally of no consequence although occasionally it is a factor in causing a rise in intraocular pressure which may require pilocarpine for control.

Harrington[11] reported rupture of the anterior hyaloid in 13 of 66 eyes studied but found that no functional loss resulted in any case.

There is thus unanimous agreement with

This paper was submitted to THE JOURNAL just prior to the publication of Irvine's study, "A newly defined vitreous syndrome following cataract surgery" (Am. J. Ophth., **36**:599, 1953), in which a number of similar observations were recorded.

Knapp's[6] conclusion that, although the importance of the zonulocapsular barrier for the health of the eye is an unsettled question, "late examination of many patients and clinical experience have not shown any evidence of deterioration of the eye from this cause" (that is, loss of the barrier).

In this study there will be presented what appears to be, therefore, the first series of cases in which postoperative rupture of the anterior hyaloid has had such damaging effect upon the eye that the occurrence of this complication can no longer be lightly regarded.

Materials and Methods

This study includes only those eyes on which there was performed uneventful intracapsular lens extraction without vitreous loss, and any case with rupture of the lens capsule, vitreous loss, or other operative complication was excluded. Most of the cases have been round-pupil intracapsular extractions with peripheral iridotomy, although a small number of selected cases had total iridectomy. The material has been drawn entirely from our private practice so that postoperative follow-up has been carefully controlled.

The operative technique consisted of dissection of a Van Lint flap, direct incision of two grooves in the exposed limbus, placement therein of two silk appositional sutures, keratome-scissors incision, peripheral iridotomy (or rarely total iridectomy), and intracapsular extraction by the forceps-hook tumbling method, although occasionally, with smooth or tense capsules, the erisophake was used.

The patients left the hospital on the fourth or fifth day and were examined (including slitlamp biomicroscopy) on the fifth to 10th postoperative day and at frequent intervals thereafter. It must be said that early in the series there was no reason to be interested in such early postoperative slitlamp examination, but in approximately the last 200 cases this has been the routine.

TABLE 1

Incidence of postoperative hyaloid rupture

	Intact Hyaloid	Ruptured Hyaloid	Percent Ruptured
Round pupil extractions	187	31	14.2
Combined extractions	29	3	9.4
Total	216	34	13.6

Observations

It may be stated at once that, of the last 250 eyes subjected to uneventful intracapsular cataract surgery, 34 have developed spontaneous postoperative rupture of the anterior hyaloid membrane. These represent truly "postoperative" ruptures for in only one case was a tear observed in the anterior hyaloid membrane at the time of the first postoperative slitlamp examination.

Of the 250 cases, 29 were combined intracapsular extractions while the remaining 221 were round-pupil intracapsular operations. The incidence of spontaneous rupture in the round-pupil group was 14.2 percent while in the total-iridectomy group it was 9.4 percent (table 1).

From the data in Table 2 it is seen that the incidence of postoperative hyaloid rupture is rare under the age of 55 years but there is no increasing incidence with advancing years.

More than half of the patients in this series have had bilateral intracapsular opera-

TABLE 2

Age incidence

Age	Total Operations	Ruptured Hyaloid	Percent Ruptured
Under 50	21	0	0
51–55	20	1	5.0
56–60	24	3	12.5
61–65	51	10	19.6
66–70	58	7	12.0
71–75	37	6	16.2
76–80	35	7	20.0
81–85	4	0	0
Total	250	34	13.6

tions but in only two patients (four eyes) has rupture of the anterior hyaloid membrane in both eyes been observed. Bilaterality of rupture in this series is, therefore, rare.

The earliest case that was observed occurred on the fifth postoperative day, the most common time for rupture was in the third postoperative week, and the latest observed in our series was six months after surgery. It is apparent, therefore, that a complication occurring in 13.6 percent of eyes subjected to uneventful intracapsular cataract extraction is worthy of emphasis.

In general the ruptures of the anterior hyaloid have been of two distinct types which may be classified as those with localized vitreous spread and those with generalized vitreous spread.

The localized ruptures are those involving one or more small breaks with the slight local herniation of small fingers of free vitreous. In these, although there are formed one or several small blebs on the convex anterior vitreous face, the main mass of the anterior vitreous body does not invade the anterior chamber but remains confined *locally* just in front of the pupillary space. In this type there has occurred no serious complication.

In the second, or generalized, type the vitreous bursts through its anterior limiting membrane and extends *generally* throughout the confines of the anterior chamber. It is in this group that all of the significant complications have been encountered. The two types appear in about equal frequency, for there were 17 cases in each category in the 34 cases of ruptured hyaloid.

In passing then to a consideration of the clinical importance of postoperative rupture of the anterior hyaloid membrane, it becomes necessary to describe in detail the findings in a number of the eyes wherein the success of the cataract operation was impaired because of pathologic changes apparently initiated by rupture of this membrane.

The complications that have been observed are (1) acute congestive glaucoma, (2) opacification of the vitreous, (3) detachment of the retina, (4) marked distortion of the pupil, ectropion uveae, and vitreous-induced changes in the chamber angle, and (5) permanent decrease in visual acuity of unexplained etiology.

1. Acute congestive glaucoma

M. C., aged 63 years, had an uncomplicated round-pupil intracapsular extraction of the right eye on December 24, 1952. Convalescence was normal and in the fourth postoperative week the anterior hyaloid was observed to be intact and the vision was correctible to 20/20. On February 3, 1953 (about the sixth postoperative week), routine examination revealed a flat anterior hyaloid, with a single tiny superior central break.

Two days later the patient experienced onset of sudden pain, redness, and decreased vision in the right eye and, when examined the following morning, vision of the right eye was down to 20/100, the globe was intensely injected (but did not present the markedly edematous congestion of the ordinary attack of acute congestive glaucoma), the cornea was steamy, the pupil was five mm., and the red fundus reflex was barely seen. The tension was 80 mm. Hg (Schiøtz).

The cornea was cleared with a drop of glycerin and slitlamp examination revealed rupture of the anterior hyaloid with generalized spread of vitreous over the entire anterior chamber, except superiorly, with many foci of vitreous-cornea contact. The fundus could now be seen quite clearly and no retinal vessel occlusion or engorgement was present, although there was seen one tiny linear superficial hemorrhage just superior-nasal to the disc.

At this point the problem was one of an attack of acute absolute glaucoma obviously induced by anterior hyaloid rupture with the so-called *generalized* vitreous spread. Since this type of acute glaucoma was previously unreported, there was great doubt as to the type of therapy to employ. The

peripheral iridotomy was open and there was no reason to believe we were dealing with a pupil-block glaucoma.

The patient was immediately hospitalized, given demerol in large doses, and placed on trial therapy with intensive miotics. In two hours the tension was still 80 mm. Hg so a retrobulbar injection of procaine-adrenalin and hyaluronidase was performed. This lowered the tension to 50 mm. Hg but in half an hour it began to increase.

Atropine and 10-percent neosynephrine therapy was then initiated but again for two hours the pressure remained at 80 mm. Hg. At this point DFP and pilocarpine were used and in two hours the tension began slowly to decrease. When the tension was 70 mm. Hg an adrenalin pack was used but it had no effect. DFP and pilocarpine were used every two hours through the night and the next morning the tension was down to 42 mm. Hg.

Tension fluctuated on this treatment between 40 and 70 mm. Hg (Schiøtz) for the succeeding two days and then suddenly decreased to 12 mm. Hg.

On discharge from the hospital one week after onset of the attack, vision in the right eye was correctible to 20/50, the globe was slightly injected, and slitlamp examination revealed the anterior chamber full of free vitreous with vitreous in corneal contact below, barely escaping contact with the endothelium centrally and with vitreous extending into the chamber angle in the inferior half.

On pilocarpine and eserine, tension of the right eye has remained controlled to the present but the vision is not correctible beyond 20/30. In this case, therefore, it appears that miotic therapy was more effective than treatment with mydriatics and/or decongestants.

2. Opacification of vitreous

D. C., aged 78 years, had an uncomplicated round-pupil intracapsular extraction of the left eye on October 25, 1950. On the 10th postoperative day the lids were flat, the globe was almost pale, and the media were crystal clear. The anterior hyaloid was intact.

On the 12th postoperative day, the lids were edematous, the globe presented a marked ciliary injection, and there was a *localized* rupture of the anterior hyaloid. On the 20th postoperative day, there was seen a *generalized* rupture with free vitreous filling the anterior chamber. Grossly there were two patches of opacified vitreous in the center of the anterior chamber and the fundus could be seen only hazily.

Since Reese[2] had been successful in reducing intact hyaloid herniation by using 10-percent neosynephrine followed by DFP, we attempted to clear the anterior chamber of the free vitreous by his method but it was not successful. The eye was kept atropinized but the vitreous in the anterior chamber became cloudier, a condition which progressed despite mydriatics, heat, and subconjunctival injections of cortisone.

Although the corrected vision in the second postoperative week had been 20/25, it gradually deteriorated with the increasing cloudiness of the free vitreous in the anterior chamber to 20/100. The visual acuity has remained at this poor level until the present, two and one-half years after the operation.

Complete examination of the eye has not revealed any cause for the poor vision other than the opacified vitreous, and the intraocular pressure has never been abnormal. Biomicroscopy at present reveals a clear cornea, a small notch in the otherwise round pupil at the 11-o'clock position, and degenerated cloudy vitreous extending throughout the anterior chamber, up over the peripheral iridotomy coloboma and into the chamber angle everywhere except temporally.

3. Detachment of the retina

E. K., aged 62 years, had an uncomplicated round-pupil lens extraction (R.E.) on November 15, 1950, and convalescence was normal until the 12th postoperative day.

On this day there were the typical findings of a rupture of the anterior hyaloid with *generalized* vitreous spread throughout the anterior chamber. There was actual visible contact with the corneal endothelium but no slitlamp sign of altered corneal metabolism appeared.

Ten days later there was a definite layer of aqueous humor separating the most anterior masses of free vitreous from the cornea and the globe began to whiten. By the third postoperative month, the corrected vision of the right eye was 20/20, the pupil was round but the vitreous was still present as free masses throughout most of the anterior chamber. However, four months after the cataract surgery, the anterior hyaloid membrane had regained a smooth and only slightly-convex contour and appeared like a normal unruptured hyaloid! The remainder of the anterior chamber was now free of vitreous.

Fifteen months after the cataract operation, routine slitlamp examination revealed a localized rupture of the anterior hyaloid and 19 months after surgery, we found again a rupture with *generalized* spread, the vitreous advancing into the chamber angle in all four quadrants (but with retention of 20/20 vision).

In the 24th postoperative month the patient suddenly noted marked decrease of vision in the right eye and examination revealed an inferior bullous detachment of the retina with a large horseshoe-shaped tear. Slitlamp examination revealed taut-drawn bands of vitreous extending from two quadrants of the anterior chamber angle to the main body of the vitreous in and behind the pupillary space.

The retinal detachment was treated surgically on November 10, 1952, with good anatomic result and improvement of vision from finger-counting to 20/70, but two months later there supervened another retinal detachment with large disinsertion. A scleral resection operation was performed but it was a failure. It is of interest to note that the chief bands of vitreous in the anterior chamber after the last rupture of the anterior hyaloid were in the superior temporal chamber angle, while the retinal tear and disinsertion were in the inferior nasal retinal periphery, suggesting the mechanism of direct traction.

Parenthetically, it may be added that in November, 1951, while the anterior hyaloid of the right eye had reformed and had a normal unruptured appearance and vision of the right eye was 20/20, an uneventful round-pupil intracapsular cataract extraction was performed on the other eye. On the ninth postoperative day, this eye developed a rupture of the anterior hyaloid of the generalized type but to date its vision is 20/20 and no serious complication has developed.

It is not being proposed that detachments of the retina following uncomplicated intracapsular cataract extraction are commonly caused by the changes resulting from postoperative rupture of the anterior hyaloid. It is believed, however, that this mechanism should be regarded as a possible cause and that careful biomicroscopic examination of eyes developing aphakic detachments should be performed with this possibility in mind.

4. Marked distortion of the pupil, ectropion uveae, and vitreous-induced changes in the chamber angle

L. B., aged 65 years, had an uneventful round-pupil intracapsular cataract extraction of the left eye on November 1, 1950. Convalescence was normal but on the 19th postoperative day there was observed a *generalized* rupture of the anterior hyaloid.

Three months later the anterior chamber was still filled with free vitreous and there was vitreous-cornea contact without corneal change. The pupil had begun to be pulled out temporally by a few denser bands of organized vitreous and these bands extended into the chamber angle.

By the sixth postoperative month the pre-

viously round pupil had been pulled in extreme degree toward the temporal chamber angle, producing a marked horizontal pear-shaped pupil. The displacement of the pupil by the vitreous was so marked that an ectropion uveae could be clearly seen grossly.

The bands of vitreous extending into the temporal chamber angle have now formed scars in the angle presumably the result of fibroblastic proliferation. The tension, however, has remained normal and the corrected vision is still 20/20. This case, then, represents anatomic rather than functional damage, although we shall be on guard for glaucoma or detachment of the retina. Notching of the pupil alone has been observed in approximately 15 other cases of anterior hyaloid rupture.

5. Permanent decrease in visual acuity of unexplained etiology

Several patients in this series have had bilateral round-pupil intracapsular cataract extraction followed by unilateral rupture of the anterior hyaloid of the generalized type. Although the vision in the affected eye is correctible to 20/25 or even 20/20, the patient is acutely aware that the vision is not crystal clear as in the fellow eye with unruptured hyaloid.

In one patient (B. H., aged 65 years) the eye with the ruptured hyaloid, with 20/20 postoperative vision, has final corrected vision of only 20/40 not improved with pinhole. In the absence of any other sign it is believed probable that this decrease is due to internal astigmatism produced by the irregularly refracting masses of free vitreous in the anterior chamber.

Cases of unusual clinical interest but without damage to the eye

L. D., aged 57 years, had right asteroid hyalitis observed prior to the development of cataract. On July 6, 1950, she had an uneventful round-pupil intracapsular cataract extraction of the right eye, had a normal convalescence, and then developed a *generalized* rupture of the anterior hyaloid in the fifth postoperative week. Within three months, a new condensation membrane formed about the vitreous far forward in the anterior chamber and the eye presented both grossly and biomicroscopically a beautiful picture of asteroid hyalitis in the anterior chamber.

B. S., aged 68 years, had bilateral acute anterior uveitis in 1947. She later developed cataracts and on March 6, 1952, an uneventful round-pupil intracapsular cataract extraction was performed on the left eye. There was mild postoperative iritis which left the anterior hyaloid membrane peppered with fine brown pigment.

Two months postoperatively there occurred a single, small, central, *localized* type rupture of the anterior hyaloid with protrusion through this of a large mushroom of vitreous. This rupture excited a mild flare-up of iritis. A new intact hyaloid membrane formed over this mushroomed vitreous and the new membrane in turn became dotted with iritic pigment.

The slitlamp today shows distinctly the location of the two concentric hyaloid membranes, the old and the new, their exact locations betrayed by the two concentric layers of pigment.

Discussion

Having indicated that postoperative rupture of the anterior hyaloid membrane may lead to serious complications, it is to be noted that such grave consequences are fortunately rare. Nevertheless, the evidence presented suggests the need for more careful biomicroscopic examination following routine intracapsular cataract extraction.

In the case of acute congestive glaucoma, there arises the theoretic possibility that the attack of glaucoma may have preceded the generalized type rupture of the anterior hyaloid membrane, but there are several facts that militate against this consideration. Two

days prior to the acute attack slitlamp examination revealed a tiny rupture in the anterior hyaloid and the eye was symptom-free. This is considered to have been the beginning of the rupturing process which quickly became extensive.

Secondly, there have been several cases of intracapsular, aphakic, noncongestive glaucoma with intact anterior hyaloid in which the tension was sufficiently high to require DFP for its control, and in none of these cases has the high tension provoked a rupture of the anterior hyaloid.

Thirdly, since the iridotomy coloboma in the present case was seen to be open, there was no reason to believe that the high intraocular pressure would act on the anterior hyaloid as a *vis a tergo,* but rather as a generalized force from both in front and behind. It is concluded, therefore, that this case represents a true instance of rupture of the anterior hyaloid membrane with generalized vitreous spread in which these changes in turn caused the acute congestive glaucoma.

It is probable that the mechanism of production of this type of glaucoma is similar to that following operative vitreous loss, the chief difference being that the filling of the anterior chamber with vitreous occurs in one type during the operation, in the other as a postoperative complication.

The present study has produced some data as to the mechanism and time of production of the ruptures in the anterior hyaloid membrane. Reese[2] has stated that "the anterior hyaloid membrane may be torn at the time of operation without loss of vitreous provided there is no vitreous pressure present." That this does not occur typically is attested to by the fact that in only one case was a rupture found in the anterior hyaloid at the time of the first slitlamp examination several days postoperatively.

Reese's suggestion of the pulling away of some of the lamellae of the anterior hyaloid membrane during intracapsular extraction does explain the predisposition of the membrane to ectasia and it is proposed that the logical next step in this process is *rupture* (usually in the third postopertaive week) of the membrane. It is only if this rupture becomes extensive, with vitreous spread generalized throughout the anterior chamber, that one need fear the complications already described.

The role of vigorous physical activity as a rupture-producing factor is to be considered. The observation of the rarity of bilateral rupture of the anterior hyaloid in bilateral intracapsular aphakia suggests that excessive physical activity is not an important factor in the production of hyaloid rupture, a view further strengthened by the fact that the majority of our patients with ruptured anterior hyaloids have been elderly people of sedentary habits (table 2). In this connection it has been considered that an important rupture-producing factor may be the simple rotary movements of the eyeball, stressed by Lindner in his development of pinhole spectacles for retinal detachment.

It would be of interest to compare the incidence of rupture of the anterior hyaloid in this series, in which the tumbling technique of intracapsular extraction has been used, with a similar series using the sliding or nontumbling method of delivery.

The question arises that postoperative wound leak may be an important factor in the production of rupture of the anterior hyaloid. The data of this study are not in agreement with this idea for in only one case of rupture was there observed the classical triad of wound leak (loss of anterior chamber, hypotony, and positive dye test), and in this case the leak preceded the first evidence of rupture by three weeks.

It appears logical to assume that the most important factor determining the preservation of the intact state of the anterior hyaloid membrane is the absence of degeneration of the vitreous and its hyaloid membrane. That there is not necessarily a correlation between the health of the vitreous and that of its

hyaloid, however, is illustrated by the following case:

In an intracapsular extraction on a highly myopic eye with known marked vitreous degeneration, as the lens tumbled it was followed by presentation of a markedly bulging but intact hyaloid. The lens was used as a tampon and the assistant carefully pulled the corneoscleral sutures taut so that there was no loss of vitreous, but it appeared certain that had the anterior hyaloid not been a membrane of integrity it would have ruptured and vitreous would have been lost. Postoperatively the anterior hyaloid was seen to be flat and intact.

This represents, therefore, an instance wherein a healthy hyaloid existed in the presence of a diseased vitreous body.

It is of interest to note that in no case has any opacification of the cornea resulting from contact with free vitreous been observed, even in two eyes in which moderate cornea guttata had been present.

Some anterior hyaloid membranes are of unusual strength, distensibility, and elasticity. One patient in this series developed an acute intestinal obstruction two days after a round-pupil intracapuslar lens extraction and, despite extreme and prolonged vomiting, maintained an intact anterior hyaloid. Another patient, a high myope, had bilateral round-pupil intracapsular lens extraction and, approximately one year after the surgery, was in a serious automobile accident. She suffered a very severe cerebral concussion but both anterior hyaloids remained intact. These cases offer further evidence that excessive physical activity or external trauma are probably not important in the pathogenesis of hyaloid rupture.

The important fact remains that, whatever may be the cause of the postoperative rupture of the anterior hyaloid membrane, once the rupture is observed its presence must invoke consideration of the complications herein described.

Summary and Conclusions

1. A study has been made of the occurrence and effects of spontaneous rupture of the anterior hyaloid membrane following uncomplicated intracapsular cataract surgery.

2. Of 250 eyes subjected to intracapsular cataract extraction, 34 eyes (14 percent) developed spontaneous postoperative rupture of the anterior hyaloid.

3. Two distinct types of rupture of the anterior hyaloid have been observed, those with *localized* vitreous spread and those with *generalized* vitreous spread throughout the anterior chamber.

4. No significant complication has been observed in the group of rupture with *localized* vitreous spread.

5. In the group of rupture with *generalized* vitreous spread there have occurred a series of pathologic changes which have seriously interfered with the anatomic or functional success of the cataract operation.

6. These pathologic changes have resulted, in the present series, in (a) acute congestive glaucoma, (b) opacification of the vitreous, (c) detachment of the retina, (d) marked distortion of the pupil with ectropion uveae and vitreous-induced changes in the chamber angle, and (e) permanent decrease in visual acuity.

7. The type of acute congestive glaucoma encountered here has been previously unreported. A method of its successful treatment is described.

8. In view of these observations postoperative rupture of the anterior hyaloid membrane is a complication that can no longer be considered innocuous.

1345 North Bayshore Drive (32).

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MACULAR EDEMA IN ASSOCIATION WITH CATARACT EXTRACTION*

JOHN V. V. NICHOLLS, M.D.
Montreal, Quebec

In the last few years several patients exhibiting edema of the macula following cataract extraction have come under my care. The coincidence of these lesions gives rise to some interesting speculations. Apparently the causal relationship is complex. A report of my cases may serve to throw some light on it.

This is the first time in my own private practice that I have noted this complication. But from the very nature of macular edema, one would expect to find it occurring frequently after cataract extraction. It is interesting to note, therefore, that even the most diligent search of the literature has failed to disclose any mention of it in this association.

I have discussed my findings with several ophthalmologists. Kirby[1] states that he has "seen it frequently after glaucoma surgery but not after cataract operation." He feels it is likely to follow any opening of the eye. Stallard[2] states that he had had two cases. MacMillan[3] and Ramsay[4] cannot remember any specific cases in their experience, but agree with Kirby that it must be common.

As a disease entity macular edema is exceedingly common and is well known to all. It has been studied by several workers. Many[5–10] believe the majority of cases to be of vascular origin. Frequently there is a familial element.[11]

In 1952, I[12] described a series of cases of macular edema of various types, and attempted to show that they form an interrelated group. Except for those cases of bacterial origin, I believe the condition is brought about by slowing of the arterial flow owing to arteriolar sclerosis, or spasm, or a combination of both conditions. This dysfunction may occur in the retinal circulation or the choroidal circulation, or both.

This view is supported by recent histologic studies by Klien.[6, 13]

I attempted to show that many of the patients so affected suffer from anxiety, and that this frequently is a precipitating cause. This opinion supported and extended those of Horniker,[14] Harrington,[15, 16] and Zeligs.[17] I found that, when the anxiety was controlled, and the patients were given vasodilators, improvement often occurred.

In my present study I am reporting seven cases of macular edema associated with cata-

* From the Departments of Ophthalmology of the Royal Victoria Hospital and McGill University. Read at the annual meeting of the Canadian Ophthalmological Society, Minaki, Ontario, June, 1953.

ract extraction. I believe that this complication is much more common than we suspect. From a study of these case reports, it is possible to elucidate the pathogenesis and prognosis of this complication, and to gain some idea of the best treatment.

Case reports

Case 1

F. H. first came under my care for cataracts in September, 1946, at the age of 82 years. At that time her vision was 6/20 (corrected) in each eye. Because of the lens opacity in each eye the fundus details could not be seen. The general health was excellent. The patient was somewhat nervous and tense at the time of operation, though emotionally she was usually relatively stable. The blood pressure on admission was 180/90 mm. Hg.

On December 1, 1949, an intracapsular cataract extraction with peripheral iridectomies at the 11- and 2-o'clock positions was carried out successfully. The wound was closed with three conjunctival sutures. On the fifth day after operation a small hyphema appeared, which rapidly disappeared in the next three days.

The patient was discharged on the 13th postoperative day with an eye that appeared very well. When checked on January 19, 1950—50 days after operation—the right eye was quiet. The pupil was round and the vitreous face intact. The fundus, except for mild arteriolar sclerosis, was quite normal.

Following refraction the vision with a +12.5D. sph. was 6/7 partial. There was no astigmatism. This correction and a reading addition of +3.0D. sph. was prescribed.

The patient was examined again on March 17, 1950—107 days after operation. The pupil of the right eye was misshapen to form a horizontal oval 5.0 by 3.0 mm. The patient stated that this had been present for about a week. There was no pain but she thought that she did not see quite so well.

Slitlamp examination showed that the vitreous had prolapsed through the pupil in a tongue-shaped mass and was adherent to the corneoscleral wound at the 12-o'clock position. The fundus appeared normal except for mild arteriolar sclerosis as mentioned above. The vision, with correction, in the right eye was 6/12 partial. The patient was given eserine drops (0.25 percent, three times daily) for the right eye.

The patient was examined 10 days later. The right vision was poorer. It was 6/18. The pupil was essentially unchanged. Through it the fundus could be seen easily. The macula now was mildly edematous and showed fine pigment mottling. There were no hemorrhages or exudates.

On April 6th—another 10 days later—the pupil was much rounder and the fundus was quite normal. The vision had improved to 6/7 partial. Two weeks later the pupil was more nearly regular and the vision was 6/6 partial. On June 8th, the pupil was round. The vitreous had returned behind the pupil and the macula was quite normal in appearance.

The patient has been seen at intervals since that time and there has been no change. The tension has been normal throughout.

Case 2

W. J. H. first came under my care for nuclear cataracts in July, 1947, at the age of 70 years. The fundus details could not be seen in either eye. He was an apparently healthy man save for quite marked hypertension. The blood pressure was 240/100 mm. Hg on admission but decreased slightly later. He was quite excitable and emotionally unstable though not neurotic. In other respects his general health was excellent.

On October 3, 1949, an intracapsular cataract extraction with two peripheral iridectomies was carried out successfully on the right eye. The wound was closed with five conjunctival sutures. The patient was discharged 14 days later, after an uneventful recovery.

When checked on November 17th—45 days after operation—the right eye was quiet. The pupil was round but displaced very slightly to the nasal side. The vitreous face was intact. The fundus examination showed very mild generalized arteriolar constriction and sclerosis. There were no hemorrhages or exudates. The macula and nervehead were quite normal.

Glasses were prescribed. With +12.0D. sph. ⊃ +0.75D. cyl. ax. 160° his right vision was 6/9 partial. When the glasses were checked a week later, the condition of the right eye was unchanged.

On January 16, 1950—105 days after operation—the patient returned stating that his vision had been blurred for several days and the pupil deformed. There were no other symptoms.

Examination showed that the pupil was grossly misshapen—it looked like a hammock pupil with the hammock up-ended vertically. The nasal half of the iris was retroverted, owing to a large prolapse of the vitreous forward through the pupil. This had become adherent to the nasal half of the corneoscleral wound.

The fundus appeared normal, except for the aforementioned arteriolar constriction and sclerosis. The vision of the right eye was 6/18. The patient was placed on eserine drops (0.25 percent, three times daily).

On February 16th, the vision had improved to 6/15 partial. On this occasion it was felt that there was some macular edema. By March 27th, the vision was 6/12 and slowly improved to 6/9 in the subsequent weeks. However, there was no change in the pupil. The tension at all times was within normal range.

Since the spring of 1950 this patient has had three attacks of blurred vision, each one lasting

several weeks. On the occasion of each of these attacks it was noticed that there was macular edema. Since the autumn of 1950, these have been treated with nicotinic acid (50 mg., three times daily before meals), and latterly with priscoline (25 mg., three times daily before meals).

Following each attack the vision has returned to 6/9. There has been no pigment change in the macula. No hemorrhages or exudates ever appeared. No further changes in the pupil or vitreous face have occurred. The tension has remained normal throughout.

CASE 3

H. M. G. first came under my care in March, 1950, for cataracts at the age of 75 years. The fundi could not be examined because of the lens opacities.

She was a healthy individual except for moderate hypertension. The blood pressure was 200/95 mm. Hg at admission but decreased slightly subsequently. The patient was very unstable emotionally. Throughout her prolonged treatment she remained a nervous, overanxious, and dependent person.

On August 20, 1951, the cataract of the left eye was removed. An intracapsular extraction with a single peripheral iridectomy was attempted. The patient lost her nerve at the crucial moment. When the lens capsule was grasped, she suddenly lifted her head causing a subluxation of the lens below. The patient was given a few minutes to recover her nerve, then a full iridectomy was performed at the 12-o'clock position and the lens was removed with a wire loop. Fortunately, only a small amount of vitreous was lost.

The wound was closed with two preplaced mattress-type corneoscleral sutures, and three conjunctival sutures. At the end of the operation the eye looked relatively well.

Two days postoperatively, the patient had a nightmare and became quite disorientated. As a result, the nasal pillar of the iris prolapsed. This was discovered next day when her dressing was changed. At once she was taken to the operating room and the prolapsed iris was excised. From thence forward healing was uneventful.

The pupil has remained slightly hammock-shaped, but not particularly pulled up. The vitreous has remained adherent to the corneoscleral wound above. Following the iris prolapse and for many weeks thereafter, there was a thin film of blood on the anterior vitreous face. Because of this the vision was hazy for many weeks, and the fundus could not be examined.

The eye progressively quietened. The blood on the surface of the vitreous cleared. By December 7th—109 days after operation—with a +9.5D. sph. ⊃ +1.0D. cyl. ax. 180°, the vision was 6/9 partial. The fundus appeared quite normal save for mild arteriolar sclerosis.

The vision remained the same until March 4th—197 days after operation—when the patient returned stating that the vision of the left eye was "fluttery," and that there was a central blindspot.

The eye appeared unchanged except that the vision was only 6/18 partial. The macular area was quite edematous, with a small round pale area in its center that had the appearance of a cyst. Slitlamp examination with a Hruby lens showed a posterior vitreous detachment but no appreciable vitreous degeneration.

The patient was instructed to take more rest and was placed on nicotinic acid (50 mg., three times daily before meals). Later priscoline (25 mg., three times daily before meals) was tried but had to be discontinued as it was not well tolerated.

There was no change in the vision for about a month, then slowly it began to improve. By August 26, 1952—one year after operation—the vision of the left eye was 6/7. The fundus showed only fine pigment mottling in the foveal region. There was no apparent old or new cyst formation in the foveal region. The patient has continued taking the nicotinic acid and there has been no further change, either in the fundus or in the appearance of the pupil and anterior vitreous.

CASE 4

J. R. B. first came under my care for nuclear cataracts in October, 1948, at the age of 62 years. She was a nervous, high-strung type of individual. Her general health was good. Her blood pressure varied between 130/90 and 125/70 mm. Hg. At one time the patient was thought to suffer from pernicious anemia; however, for the six years previous to operation her hemogram had remained normal without treatment. Fundus examination could not be carried out owing to the lenticular opacities.

On October 17, 1951, an intracapsular cataract extraction with two peripheral iridectomies was carried out successfully on the right eye. The wound was closed with a single preplaced mattress-type corneoscleral suture. The conjunctiva was closed with three sutures. The patient made an uneventful recovery and was discharged on the 13th day after operation.

The right vision was slow to recover. On December 6, 1951, with +11.0D. sph. ⊃ +2.0D. cyl. ax. 170°, it was 6/18 partial.

Fundus examination disclosed mild generalized arteriolar constriction and sclerosis. There was a small patch of waxy exudate just above and to the nasal side of the foveal region. There were no other exudates, or hemorrhages. The foveal region was slightly edematous so the pit could not be discerned.

The patient was placed on priscoline (25 mg., three times daily before meals). She did not tolerate this very well, but persisted with it. On January 18, 1952—93 days after operation—the vision had improved to 6/6 partial. On this occasion the waxy exudate had disappeared and the foveal pit

could be seen quite clearly. There were no pigmentary changes about it. Hence the priscoline was stopped.

There has been no recurrence of her trouble. However, since this time there has been a gradually increasing tesselation of the whole fundus, and a number of small drusen have appeared in the macula.

On November 24, 1952, an intracapsular cataract extraction with round pupil was carried out successfully on the left eye by essentially the same procedure as that used on the right eye. This time the patient was much more composed. On admission her blood pressure was 175/95 mm. Hg which was higher than before.

Operation was carried out under local anesthetic and deep sedation. After operation she was given phenobarbital (0.5 gr., three times daily before meals). She made an uneventful recovery.

By December 19th—25 days later—the left vision with a +10.5D. sph. ⊃ +1.0D. cyl. ax. 140° was 6/9.

There was no evidence of edema in either macula. Each fundus showed considerable tesselation. The right macula showed several small drusen. The left macula appeared normal; there were no drusen.

On January 21st—58 days after operation—the vision of the left eye, with correction, was 6/12. But now the macular area of the left eye showed a change. It was slightly edematous and flecked with small pale-yellow fuzzy punctate spots. Vision of the right eye, the first eye to be operated on, remained at 6/6 and the fundus was as already described. She was given no treatment other than being told to be quiet.

By April 21st, the vision of the left eye had returned to 6/6. The right vision remained at 6/6. In the left macula the edema had subsided completely leaving several yellowish-white punctate spots which now appeared to be drusen.

At no time in either eye of this patient was there any deformity of the vitreous face or pupil. The posterior vitreous of both eyes has been normal in appearance.

Case 5

J. F. M. first came under my care in February, 1952, for nuclear cataracts at the age of 81 years. He was a relatively healthy male of a nervous disposition. He recently had lost his wife. This with his failing vision was causing him considerable anxiety so that he was not sleeping well. His blood pressure was 170/78 mm. Hg on admission, and decreased slightly later. His general health was good except for considerable arteriosclerosis.

On March 31, 1952, an intracapsular cataract extraction with a single peripheral iridectomy was carried out successfully on the right eye. The wound was closed with one preplaced mattress-type corneoscleral suture and five conjunctival sutures. Save for a small hyphema, which developed on the fifth day after operation, recovery was uneventful. The blood was slow to absorb however.

On April 22nd, the hyphema was still present. The pupil was round and the anterior vitreous face was intact, and remained so. The vitreous was so hazy that the fundus could not be seen. The vision was very blurred. By June 2nd—63 days after operation—the hyphema and the vitreous had cleared so the fundus could be seen. There was moderate arteriolar sclerosis. The macula showed edema and stippled pigmentation.

Vision of his right eye with a +11.0D. sph. ⊃ +2.0D. cyl. ax. 10° was 6/18. He was placed on priscoline (25 mg., three times daily before meals). Three weeks later the vision had improved to 6/12 and has remained unchanged since.

The macular edema slowly subsided leaving stippled pigmentation. The patient has continued taking his priscoline until very recently and there has been no further change. A very minute central scotoma has persisted.

Case 6

F. W. R. first came under my care for a cataract of the left eye with complicating myopia at the age of 79 years. Previously a cataract had been successfully removed from the right eye by Dr. J. A. MacMillan.

This eye with a correction of +1.0D. sph. ⊃ +1.0D. cyl. ax. 15° had 6/6 vision. In it there was a large myopic crescent with a large area of myopic degeneration extending from the nervehead half way to the fovea. The fovea appeared to be intact. There was mild generalized arteriolar sclerosis.

The left eye obviously was highly myopic also but the fundus could not be seen.

The general health was excellent. The blood pressure varied between 110/70 and 148/70 mm. Hg. Temperamentally the patient was an excellent type.

On May 12, 1952, an intracapsular cataract extraction with round pupil and a single iridotomy at the 12-o'clock position was carried out successfully. The wound was closed with a single preplaced mattress-type corneoscleral suture and five conjunctival sutures.

Thirteen days after operation she was discharged from hospital. The vitreous face was intact. The pupil was not quite round as there was a small iris retroversion at the 12-o'clock position but it was not pulled up. The fundus could be seen well.

There was a large myopic crescent and a large area of myopic degeneration extending from the disc almost up to the fovea. The foveal pit could not be seen. There were no hemorrhages or exudates. On June 8th—37 days after operation—the left eye was quiet and its vision with a +2.0D. cyl. ax. 180° was only 6/120.

On this occasion it was seen that the retina of the foveal region and those parts adjacent to it were edematous. There were no hemorrhages or exudates. The patient was placed on nicotinic-

TABLE 1

ESSENTIAL CHARACTERISTICS OF PATIENTS SUFFERING FROM MACULAR EDEMA FOLLOWING CATARACT EXTRACTION

Name	Age at Operation	Sex	Blood Pressure (mm. Hg)	Preoperative Complications	Operative Complications	Postoperative Complications	Date of Latter (days)	Date Onset of Macular Edema (days)	Possible Etiology	Final Vision
(1) F.H.	86	F	180/90	Art. scl.	Nil	Hyphema Prolapse vit.	5 100	117	Art. sclerosis? Cataract ext. with vit. prolapse.	6/6 pt.
(2) W.F.H.	72	M	240/100	Art. scl. hyper.	Nil	Prolapse vit.	100	105 (possibly) 135 (certain)	Art. sclerosis, vasospasm, cataract ext. with vit. prolapse.	6/9 to 6/12
(3) H.M.G.	76	F	200/95	Art. scl.	Loss vit.	Prolapse iris, hyphema	2	197	Art. sclerosis, vasospasm, complicated cataract ext. with prolapse of vitreous.	6/7
(4) J.R.B.(OD)	65	F	125/70 to 130/90	Nil	Nil	Nil	—	Unknown	Vasospasm, cataract extraction.	6/6
J.R.B.(OS)	66	F	175/95	Nil	Nil	Nil	—	58	Vasospasm, cataract extraction.	6/6
(5) J.R.M.	81	M	170/78	Art. scl.	Nil	Hyphema	5	Unknown	Art. sclerosis, vasospasm, cataract extraction.	6/12
(6) F.W.R.	79	F	110/70 to 148/70	Art. scl. Myopia	Nil	Nil	—	Unknown	Art. sclerosis, vasospasm, cataract extraction, myopia.	6/18

acid tablets (50 mg., three times daily before meals). She did not tolerate priscoline well.

By October 20th, the left vision had improved to 6/30 but there was metamorphopsia. The edema of the foveal region had decreased. In December, the macular edema was still present and now there was a small patch of pigment up and in from the foveal region. Vision with correction was 6/20. The nicotinic acid was continued.

By January 16, 1953, the vision was 6/18 but the appearance of the macula was unchanged. When last examined in April, 1953, there was no further change.

For easy reference, the salient features of these case reports have been condensed and are presented in Table 1.

DISCUSSION

It will be seen from Table 1 that there is more than one possible cause for the macular edema in most of these patients. Cases 4, 5, and 6 are the most easily explained and therefore will be disposed of first.

As already pointed out, I believe that macular edema per se in most cases is of vascular origin.[12] The disturbance is due apparently to slowing of the arterial flow which may be brought about either by arteriolar sclerosis, or arteriospasm, or a combination of both these factors. This is the basic, underlying situation. But in many cases the precipitating factor is anxiety. I believe Cases 4 and 5 can be accounted for best on this basis.

Both patients approached operation with great trepidation. It is because of this emotional disturbance that the occurrence of macular edema was associated in time with the cataract extraction. I do not believe that there was any closer relationship between the operation and the occurrence of the macular edema than this. In the patient (Case 4), there was less arteriolar sclerosis than in the patient (Case 5), hence the response to vasodilatation was better.

It is interesting to note that in Case 4, the macular edema occurring in the left eye was different from that in the right. In the left it had the typical appearance of central angiospastic retinopathy.[13] In the right the picture was not typical of this condition and there was a patch of waxy exudate.

In the patient in Case 6, there was no emotional disturbance, but there was a high degree of myopia with secondary retinal degeneration. I believe that myopic retinal degeneration in many cases also can be explained best on a vascular basis, in that the enlargement of the posterior segment of the sclera produces a relative ischemia. Further, this last patient (Case 6) had mild arteriolar

sclerosis. The operative trauma, acting in an area having a critical blood supply, precipitated the edema. The response to vasodilatation, while definite, was slow and recovery was not complete.

With the exception of the left eye of the patient in Case 4, the common feature among these patients was that the exact moment of onset of the macular edema could not be determined since it occurred so near to the time of operation. Indeed, it may have occurred just before or just after operation.

The examination of the posterior vitreous with the Hruby lens showed no disturbance in the patients (Case 4 and Case 5), while in the patient (Case 6) there was slight detachment of the posterior vitreous and some mild vitreous degeneration. With the exception of the last patient (6) and the left eye of the patient (Case 4), unfortunately this examination of the posterior vitreous was not possible until some time after operation.

The first three patients give rise to much more interesting speculation. There were a number of common features among these patients. They all had high blood pressure. The macular edema occurred only some time after operation.

In all there was prolapse of the anterior vitreous. In the first two patients, the prolapse occurred some time after operation; it occurred at about the same time as the macular edema. The exact time relationship of these was impossible to determine. The second and third patients were anxious and under great nervous tension at the time of operation. In the first patient these features while present were not severe.

In all there was a detachment of the posterior vitreous but with no vitreous degeneration. Unfortunately, I was unable to determine the exact moment at which this vitreous detachment occurred, as I did not obtain my Hruby lens until some time after the operations were carried out.

I believe also that in these three patients, vascular dysfunction was the underlying cause of the macular edema. Indeed, in the patient (Case 3), it may have been the only cause. This last patient was a most anxious and dependent type. Owing to complications at the time of her operation she had a prolonged convalescence with consequent prolongation and intensification of her anxiety status.

In the patients (Cases 1 and 2), one cannot help but speculate on the relationship of the vitreous prolapse to the occurrence of the macular edema.

Which was the cause and which was the effect?

The prolapse could possibly have played either role. Since there was no evidence of damage to the vitreous immediately after operation, and since there was absolutely no evidence of trauma postoperatively, possibly it was the effect and not the cause. Perhaps the sequence of events was: macular edema with the collection of preretinal transudate which resulted in a separation of the vitreous and the forcing forward of it to cause the anterior hyaloid membrane to break at some weakened point. It is difficult to believe, though, that the transudate would develop enough pressure to do this.

Harrington[18] in a recent paper has given an excellent description of the changes in the vitreous face following cataract extraction. He has shown that the vitreous face undergoes great change in even so-called normal patients.

It is possible in the present patients, that some distortion or some traction was produced in the anterior vitreous by the healing process, such that the slightest trauma caused a break in the hyaloid membrane, permitting the vitreous to come forward into the anterior chamber. Possibly this moving forward of the vitreous permitted a separation of the posterior vitreous. Possibly the detachment of the posterior vitreous was the precipitating factor in the production of macular edema. But the predisposing factor was a vascular dysfunction of the posterior

retina and choroid. To me, this is the most acceptable hypothesis.

I have discussed these findings with many of my confreres. It has been suggested that the macular edema may have been purely of traumatic origin, occurring either as a result of operative injury or of a postoperative blow, being of the nature of a commotio retinae.

All these patients were questioned carefully regarding the possibility of injury. They all are responsible individuals and I feel they must be believed when they say that there was no such injury.

Injury at the time of operation cannot be ruled out. But it certainly could not have been the cause in the first three patients where the onset of macular edema came a long time after operation. It has been suggested that postoperative iritis may have been the cause. All these patients were followed closely and at no time was there any evidence of any uveitis.

As I was completing the final draft of this report, a paper by Irvine[10] appeared, on what he has described for the first time as the "vitreous syndrome." In this condition, sometime following cataract extraction, the vitreous shrinks and then prolapses forward and becomes adherent to the wound. Associated with this, the eye develops recurrent spells of irritability and macular degeneration.

My first two cases, and possibly the third, would fit into this category very well, though the irritability feature was absent and the vitreous shrinkage and macular degeneration were not particularly marked.

Irvine does not mention vascular dysfunction as a predisposing factor. All my patients had hypertension, arteriosclerosis, and anxiety. I should like to add this factor, as a predisposing one, to the syndrome he describes so well.

In this study the most important point of all, and the one which has the most practical significance, is that the recovery was relatively good in all the patients. It is impossible to evaluate fully the value of vasodilating drugs. Certainly they do no harm, and by analogy with the results of this treatment in patients with macular edema not associated with cataract extraction,[12] I believe they are of some value.

Summary and Conclusions

1. Seven cases, in six patients, of macula edema in association with cataract extraction are presented.

2. In three cases, the disturbance occurred quite some time after operation and was associated with prolapse forward of the vitreous.

3. In four cases, the disturbance occurred at about the time of operation.

4. It is suggested that the predisposing cause was a vascular dysfunction of the retina and/or choroid in the macular region.

5. It is suggested that the precipitating cause in some cases was anxiety, while in others a prolapse of the vitreous through the pupil played a part.

1414 Drummond Street.

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COMPLICATIONS FOLLOWING CATARACT EXTRACTION*

With emphasis on pathologic documentation

Joseph A. C. Wadsworth, M.D.
New York

Forty years ago Dr. D. W. Greene[1] presented a paper before the Chicago Ophthalmological Society on "Complications and after-treatment following cataract extraction." Apparently some of the same complications arise today that troubled the surgeons 40 years ago. However, with the increased popularity of the intracapsular extraction, many of the troublesome complications encountered in Dr. Greene's series have been eliminated.

His introductory sentence is very important and far-reaching, and may I quote? —"Beginning with the preparation of the patient there is no step in the cataract operation that may not cause complications and require after-treatment."

This author also criticized the speculum that was used at that time because it often manifested undue pressure on the globe. All of his sections were knife sections and many of the complications were peculiar to the knife section.

I shall attempt to demonstrate some of the complications following cataract extraction and document these changes by microscopic sections when possible. One could label this discussion "Paradise Lost"; but a prompt and successful treatment of these complications might be labeled "Paradise Regained." However, a prevention of these complications would be simply "Paradise."

Infection

The first complication is infection. The early infections are most probably *ectogenous* and result in an endophthalmitis or even a panophthalmitis. Before the advent of the present antibiotics the affected eyes were usually lost; certainly lost as a visual organ. Oftentimes, it was necessary to enucleate or eviscerate following such an infection. The existence of pathogenic bacteria in the conjunctiva or lids prior to operation is often the cause for these early infections.

Usually on the second or third day there is evidence of a purulent material around the sutures. Following this, the anterior chamber becomes cloudy and filled with a purulent exudate. Later, a vitreous abscess may develop. If the condition cannot be controlled,

* From the Institute of Ophthalmology, Presbyterian Hospital. Presented before the Chicago Ophthalmological Society, February 20, 1952.

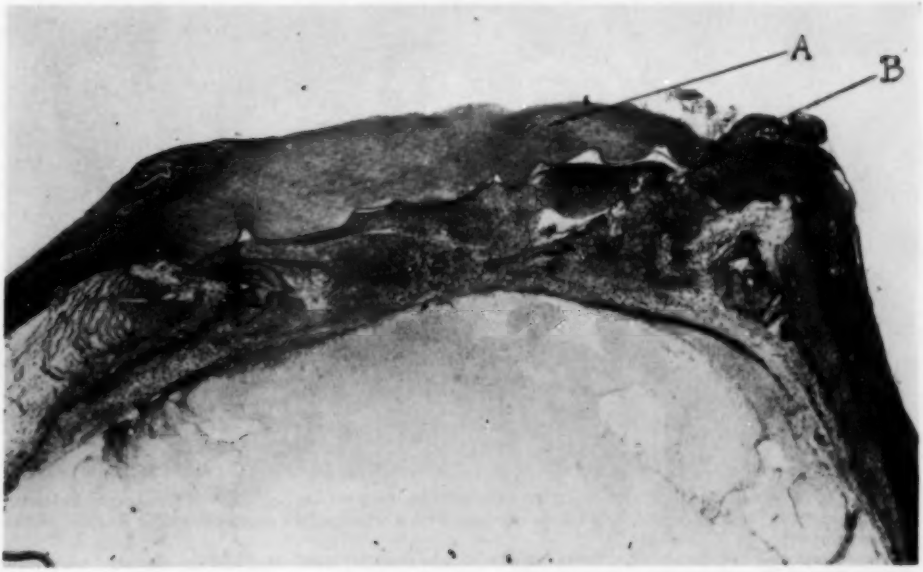


Fig. 1 (Wadsworth). A cataract extraction was performed one month prior to enucleation. A severe inflammatory reaction developed during the first week postoperatively. The anterior chamber became filled with an exudate and the cornea also lost its transparency. The eye became soft, blind, and painful, and was enucleated.

The pathologic specimen shows a disastrous inflammatory process in the eye. The cornea is edematous and the epithelium has been completely denuded. There is cellular infiltration in the cornea and at one point there is evidence of necrosis of the corneal stroma (A). The cataract incision shows poor healing and the wound is filled with fibrin (B).

The anterior chamber is completely filled with polymorphonuclear exudate where there is considerable necrosis. The exudate extends into the vitreous and involves the iris and ciliary body. The vitreous is detached and shrunken.

The eye was lost because infection had gained access to the interior of the eye through a poorly approximated wound. There was no record of cultures having been done prior to the cataract extraction.

the eye will be lost. In spite of treatment there may be sufficient changes in the vitreous to cause its shrinkage with a resultant detachment of the retina or a dense secondary membrane.

Bacteria may be introduced into the anterior chamber during the operation. The infection will then present itself as a clouding of the vitreous and anterior chamber. The end result will be the same as the above, but the onset is often sooner and the course is more fulminating (fig. 1).

Endogenous infections may come from a focus of infection somewhere else in the body. The onset is late in the postoperative course, two weeks or even months after the operation. The development of a *uveitis* during the postoperative course is often the exacerbation of an old quiescent uveitis. This does not necessarily result in a permanent loss of vision; however, glaucoma is a frequent sequela as in any uveitis (Knapp[2]).

If the operation is an extracapsular extraction, planned or not, there may be sensitivity to the lens protein and a condition of phacoanaphylactia will result. This is usually a severe type of uveitis that ends in the loss of the eye from this uncontrolled intraocular inflammation (fig. 2).

Finally, *sympathetic ophthalmia* should be

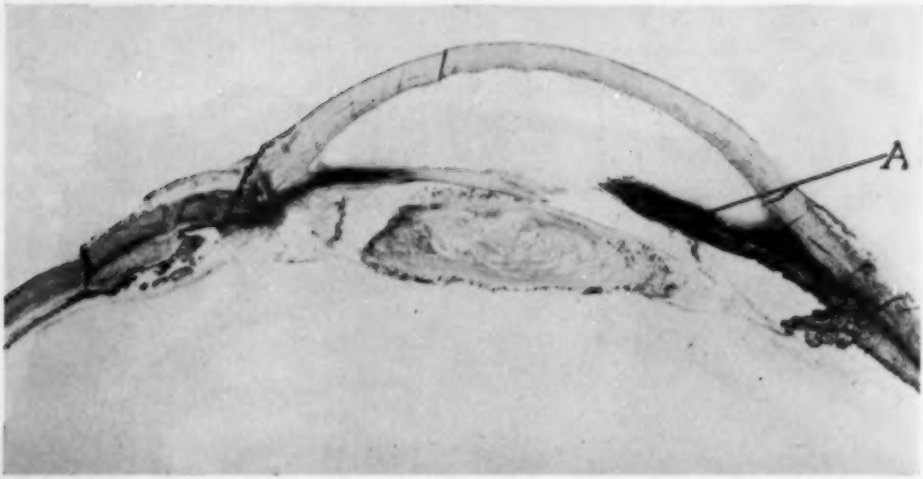


Fig. 2 (Wadsworth). An eye following an extracapsular cataract extraction. A severe uveitis developed and the anterior chamber became filled with exudate. Vision was lost, the eye was painful, and enucleation was performed.

The microscopic section shows a large amount of lens material remaining, and capsule is present in the wound. A heavy cellular infiltration is present in the anterior portion of the uveal tract (A).

The anterior chamber contains many cells, the great majority of which are eosinophils. The cellular response extends into the vitreous with cellular infiltration in the choroid.

This is a case of an extracapsular extraction with late inflammatory response. The eosinophilic type of response is highly suggestive of an endophthalmitis due to the sensitivity to lens material.

considered among the inflammations. This, fortunately, is rare, but when it does occur, the eye is usually lost. Most frequently, however, the appearance of sympathetic ophthalmia follows incarceration or prolapse of uveal tissue and it may follow a secondary procedure on a prolapse of the iris (Morgan[3]) (figs. 3a and 3b).

Prolapse of the Iris

Iris prolapse can be divided into two types. One is a small, nonprogressive type—this type may remain stationary, not increase in size, and cause no real complication. There is no indicative treatment in such cases. On the other hand, the progressive type of iris prolapse demands immediate treatment. The best treatment for such a condition is excision of the prolapsed iris and resuture of the wound (Berens[4]) (figs. 4a and 4b).

The mechanics of these two types of iris prolapse are entirely different:

The first, or nonprogressive type, if excised, causes no loss of the anterior chamber and usually no aqueous is lost. In this case it is possible that a small portion of the vitreous has pushed the iris into the wound with no further development of prolapse.

On the other hand, the progressive type, if excised, is followed by the escape of considerable aqueous followed by a loss of the anterior chamber. The latter type of prolapse is progressive and treatment is necessary as soon as the condition is recognized. A small prolapse which is nonprogressive and covered with conjunctiva may need no further treatment (Reese[5]).

Corneal Changes

The cornea may be the seat of several complications. The early loss of transparency from trauma to the endothelium while de-

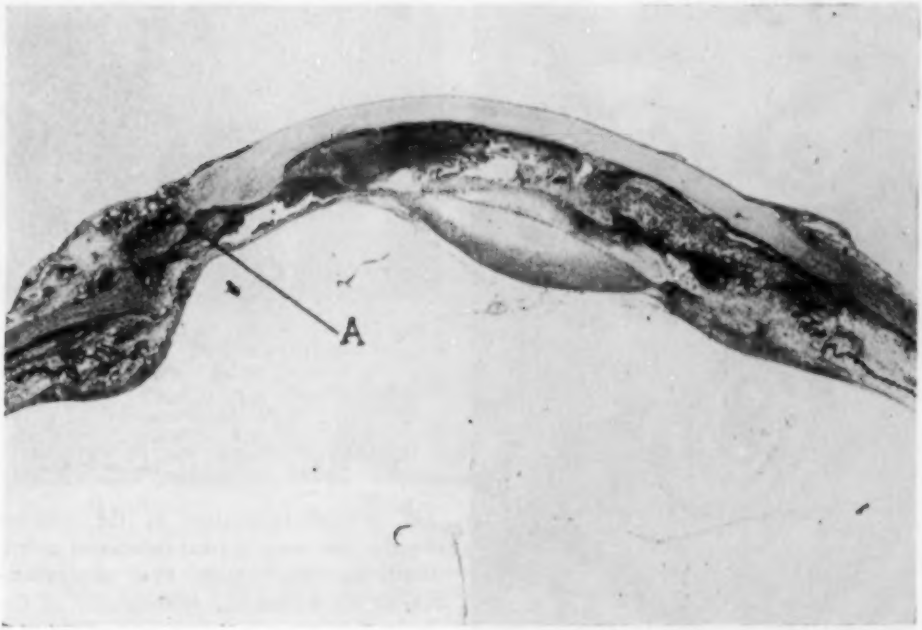


Fig. 3a (Wadsworth). This is an extracapsular cataract extraction. The postoperative course was uneventful and the eye became quiet. Cortical material persisted in the anterior chamber but with very little inflammation until approximately six weeks following the operation when the fellow eye became inflamed and photophobic with evidence of cells in the anterior chamber and deposits on the posterior surface of the cornea. Because of the diagnosis of sympathetic ophthalmia the eye was removed.

Microscopic sections show the wound to be poorly healed (A). There is inflammation both in the limbal tissue as well as the corresponding intraocular portion. The iris and ciliary body are heavily infiltrated with lymphocytes and the choroid is markedly thickened with numerous epithelioid cells and lymphocytes. The optic nerve also shows inflammation which is probably due to the inflammatory process in the anterior portion of the eye.

This is an example of a sympathetic ophthalmia following a cataract extraction.

livering the lens will usually clear by the fourth day and cause no sequelae.

If the hyaloid is in contact with the posterior surface of the cornea, changes in the endothelium take place and a striate type of keratitis will result. Disagreeable symptoms such as tearing, photophobia, and foreign-body sensation are prevalent when hyaloid is in contact with the cornea. Such discomfort to the patient may result in squeezing and restlessness with obvious complications.

If the hyaloid is separted from its apposition to the cornea by a retraction from the anterior chamber, the cornea will clear rapidly. However, if the hyaloid remains in contact with the cornea for more than two weeks, permanent changes take place in the cornea (Reese[6]) (figs. 5, 6a, 6b, and 7).

Detachment of Descemet's membrane

Detachment of Descemet's membrane can be caused by the introduction of a sharp instrument into the anterior chamber at the time of operation. This causes a small portion of Descemet's membrane to curl on itself. Later, the cornea overlying the area of the detached Descemet's membrane will show a permanent opacity (figs. 8 and 9).

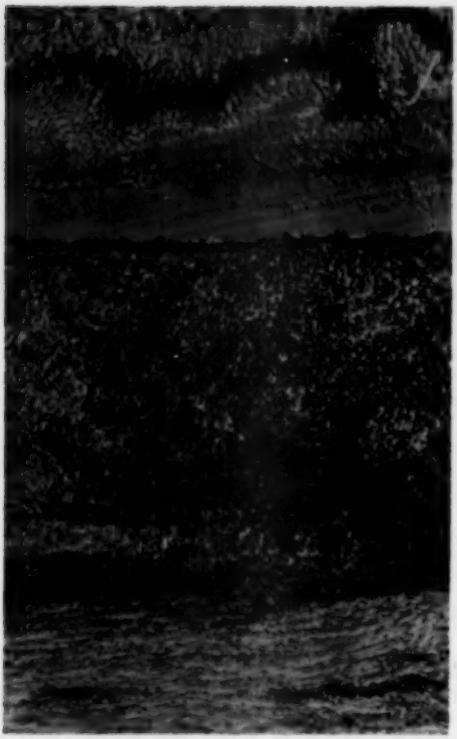


Fig. 3b (Wadsworth). The section of the choroid under high power shows heavy lymphocytic infiltration with a large proportion of epithelioid cells typical of sympathetic ophthalmia.

Fig. 4a (Wadsworth). A massive prolapse of the iris with a rupture of the wound. The patient was awakened from a sound sleep by pain in the eye. When seen the following morning there was a small knuckle of iris in the wound at the 11-o'clock position.

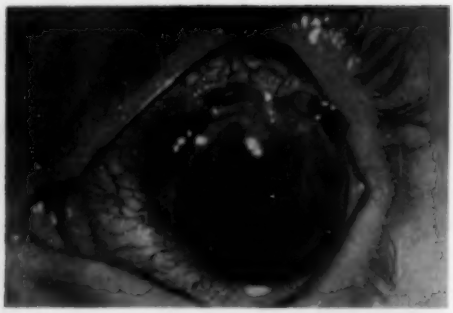


Fig. 4b (Wadsworth). By the following day the prolapse had progressed. The sutures have given way and the entire wound is gaping except at the 12-o'clock position.

Delayed restoration of the anterior chamber

A delayed restoration of the anterior chamber can cause a great number of rather disastrous complications. Poor approximation of the wound and epithelization of the anterior chamber are so closely related to a delay in the restoration of the anterior chamber that these three complications should be discussed together.

The corneal opacities due to the contact of the vitreous to the posterior surface of the cornea have already been discussed. These opacities will clear if the anterior chamber is restored, and little permanent damage is done to the cornea.

Peripheral anterior synechias may form if the anterior chamber remains flat for too long. Consequently, a secondary glaucoma will result (Puntenney[7]).

It is extremely rare to find a case of epithelization of the anterior chamber that does not have a history of delayed restoration of the anterior chamber. As a matter of fact, students of this condition believe this factor to be necessary before epithelization develops (Calhoun[8]).

Some time during the operative procedure a small portion of epithelium may be implanted into the iris. The epithelium may grow as long as the intraocular pressure is

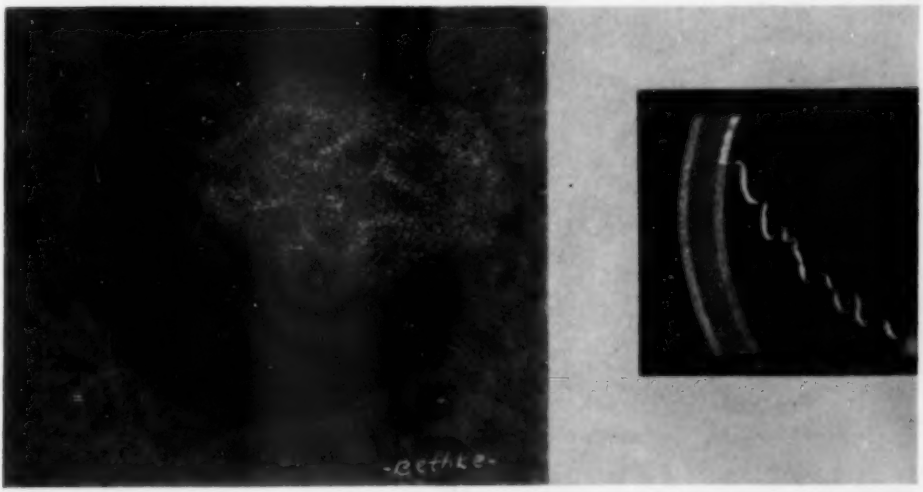


Fig. 5 (Wadsworth). A central opacity of the cornea. This opacity was present at the first dressing even though the anterior chamber was of normal depth. The anterior hyaloid remained in contact with the posterior surface of the cornea during the first 12 days. These changes represent the condition on the 12th postoperative day where the hyaloid has remained in contact with the cornea. The condition is reparable at this stage if the hyaloid can be separated from the cornea. (Reese[6].)

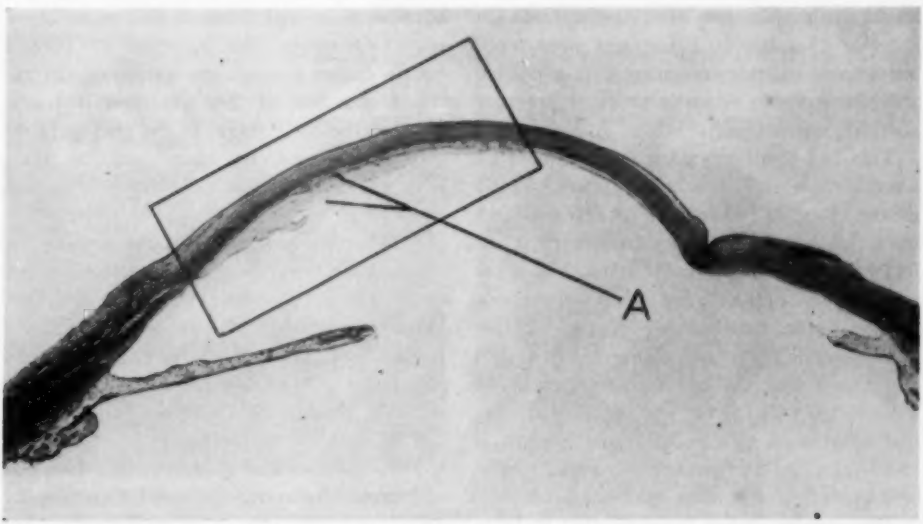


Fig. 6a (Wadsworth). This is a case in which there was a herniation of the vitreous that remained in contact with the cornea for about six weeks. A permanent opacity developed in the central portion of the cornea. The eye remained irritable and was enucleated. In the central portion of the cornea there is a reduplication of Descemet's membrane (A).

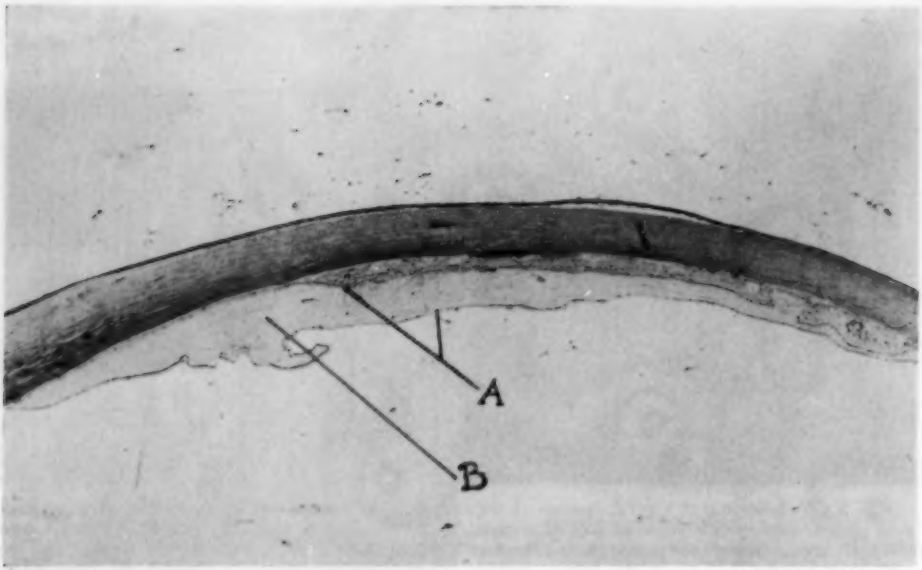


Fig. 6b (Wadsworth). High-power view of area indicated in Figure 6a. Reduplication of Descemet's membrane is clearly outlined (A). The space between the two membranes is filled with a granular substance (B).

low and the anterior chamber is shallow. No doubt the implantation of epithelium into the anterior chamber is a frequent occurrence but growth is not encouraged in a normal anterior chamber when intraocular pressure is within normal limits.

This has been somewhat substantiated by Greene's[9] work with the implantation of cancer tissue in the anterior chamber of animals. He has found that, in the normal anterior chamber, malignant tissue will grow without difficulty. However, normal or benign tissue transplanted in the anterior chamber will grow only with great difficulty. Therefore, a normal anterior chamber is not a fertile ground for the implantation of normal epithelium unless the tissue growth is encouraged by the presence of an abnormally soft eye (figs. 10a, 10b, 11a, and 11b).

The simplest cause for a delayed restoration of the anterior chamber is a suture that has been placed too deeply in the cornea. Occasionally, a through and through suture in the cornea may act as a wick and permit aqueous to escape through this small opening. Oftentimes, the removal of such a suture causes prompt restoration of the anterior chamber. If this condition is recognized in the early stages of the postoperative course little or no harm will be done (Kettesy,[10] Dunnington,[11] Dunnington and Regan[11a]).

In poor approximation of the incision the entire wound may finally heal except for one small area where there is persistent filtration. Without this filtering point, glaucoma would result. The filtering cicatrix controls the tension as in a filtration operation for glaucoma (figs. 12, 13, 14a, 14b, 15a, 15b, 16, 17a, 17b, 18, 19, 20a, and 20b).

It is obvious that glaucoma is a frequent end result following the conditions already mentioned. However, glaucoma itself is not a complication of cataract extraction, but merely secondary to some other mishap in the process of removing the cataractous lens.

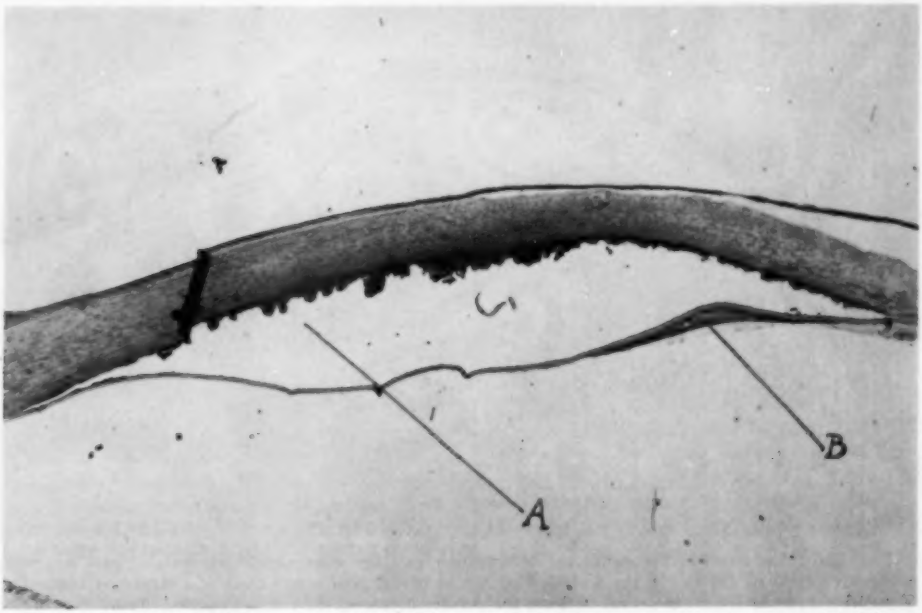


Fig. 7 (Wadsworth). This case has a similar history as the previous case except that one month following the onset of this condition a long spatula was inserted into the anterior chamber and attempts were made to separate the vitreous from its contact with the cornea. This was done satisfactorily but the central opacity of the cornea remained.

A secondary glaucoma developed; the eye became painful because of repeated abrasions of the corneal epithelium and sudden severe pain was experienced in the eye. Because of pain and loss of vision the eye was enucleated.

Examination showed hemorrhage into the cornea, separating Descemet's membrane (A). On the posterior surface of Descemet's membrane is a tissue containing pigment granules but resembling fibrous tissue (B). This was the point at which the vitreous was adherent to the posterior surface of the cornea. Contracture of the fibrous tissue caused a detachment of Descemet's membrane, resulting in a rupture of small blood vessels that had extended into the cornea.

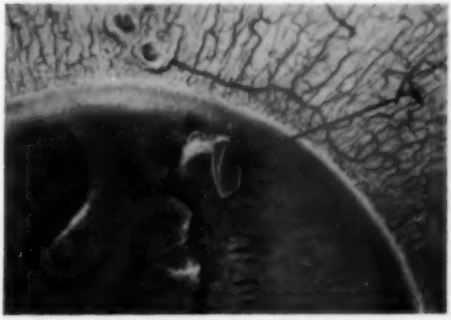


Fig. 8 (Wadsworth). A small detachment of Descemet's membrane that has been caused by the introduction of a spatula after an iridectomy (A). The detached Descemet's membrane has curled on itself, resembling a piece of cellophane in the anterior chamber.

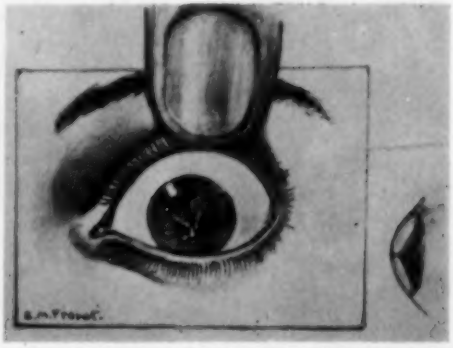


Fig. 9 (Wadsworth). This drawing shows the iris to be adherent to the cornea at a point where the tip of the keratome injured the cornea. The iris is adherent to the point of perforation and a dense opacity is present at the injured area.

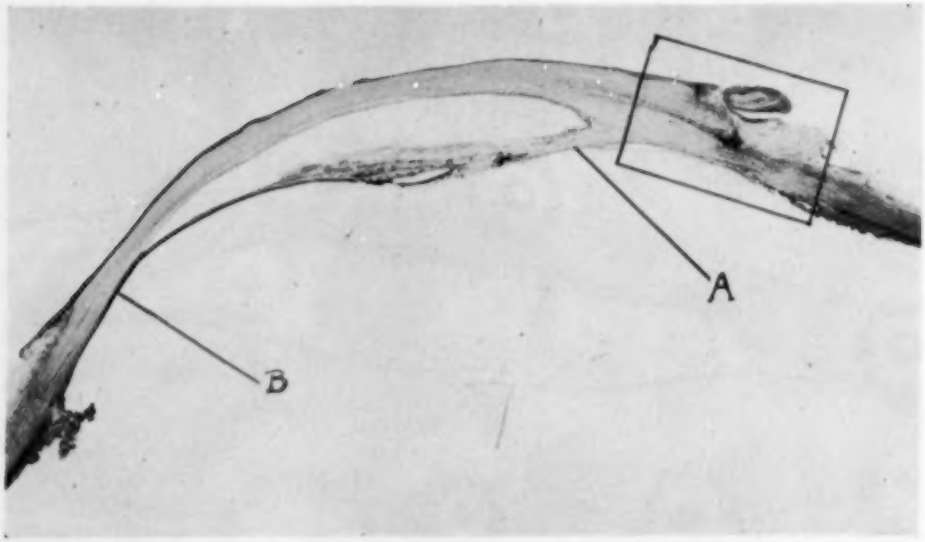


Fig. 10a (Wadsworth). Following an intracapsular cataract extraction there was vitreous loss with an incarceration of the iris in the wound. The wound healed poorly and there is a strand of connective tissue that extends from the wound bridging the pupil and causing the iris to be pulled toward the corneal incision (A). The anterior chamber did not form until late in the postoperative course and there is a wide anterior synechia with a complete obliteration of the filtration angle (B).

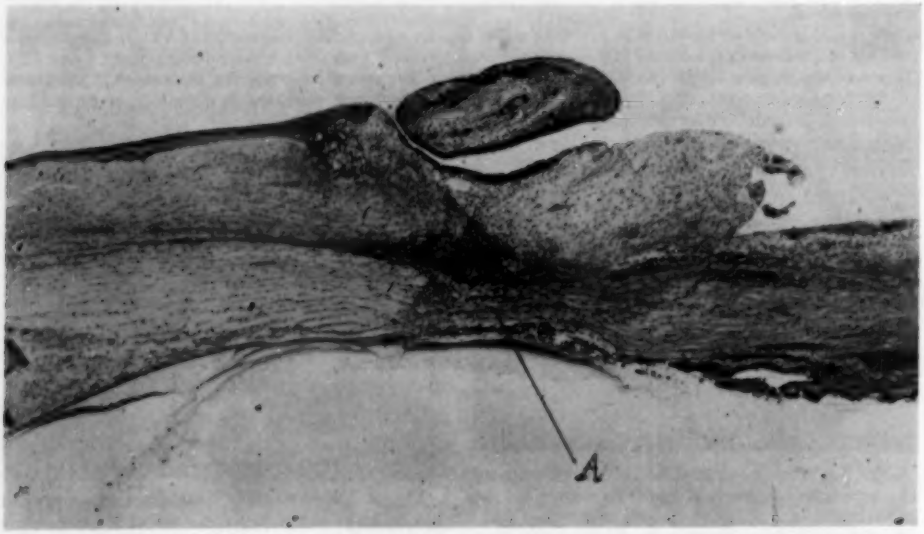


Fig. 10b (Wadsworth). High-power view of area outlined in Figure 10a. The wound shows poor approximation with fibrous tissue extending across the anterior chamber (A).

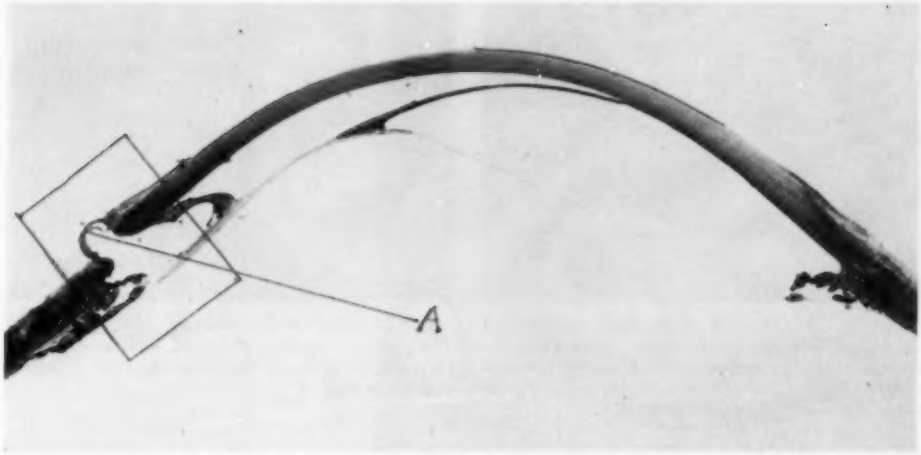


Fig. 11a (Wadsworth). The iris has prolapsed in the corneal incision and very little scar tissue has formed to support the wound (A). A staphyloma has resulted. The anterior chamber failed to reform following the operation and anterior synechias developed. The vitreous is contained in the staphyloma. Later there was epithelization of the anterior chamber.

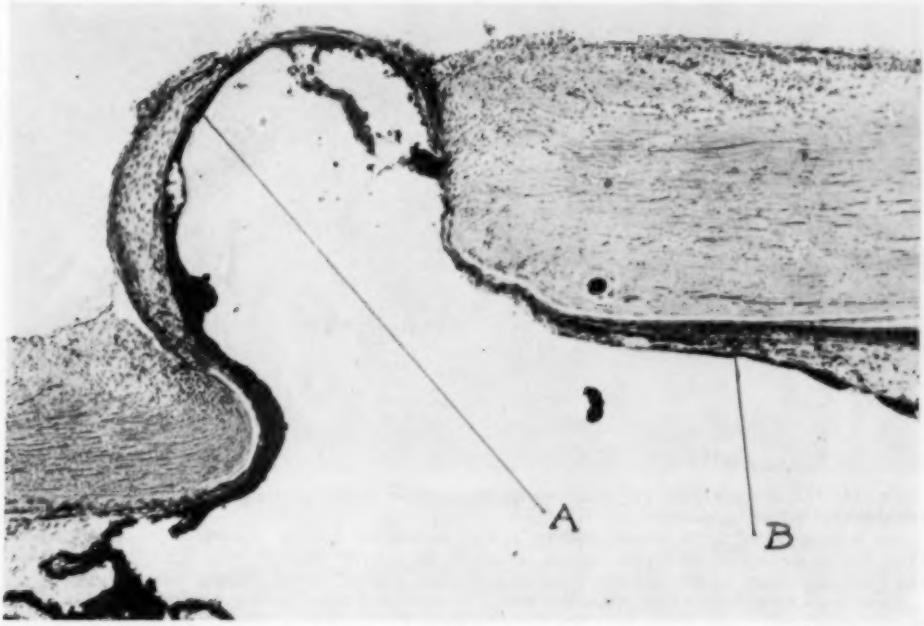


Fig. 11b (Wadsworth). High magnification of the area outlined in Figure 11a. Iris tissue bridges the wound resulting in a staphyloma (A). Epithelium has extended onto the anterior surface of the iris (B).

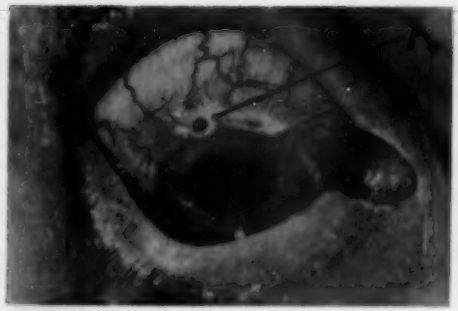


Fig. 12 (Wadsworth). A small filtering area in the wound. The anterior chamber remained flat for about two weeks after operation and slowly became restored to its normal depth, but there later developed a small filtering cicatrix (A).

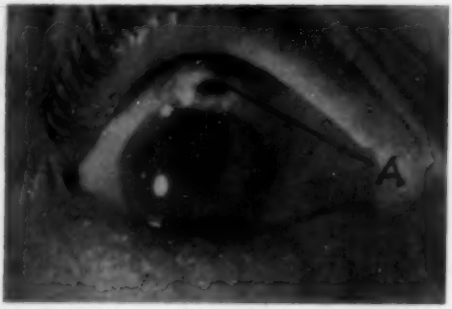


Fig. 13 (Wadsworth). A small iris prolapse (A). The anterior chamber remained shallow for some time but gradually became restored. The filtering cicatrix developed.

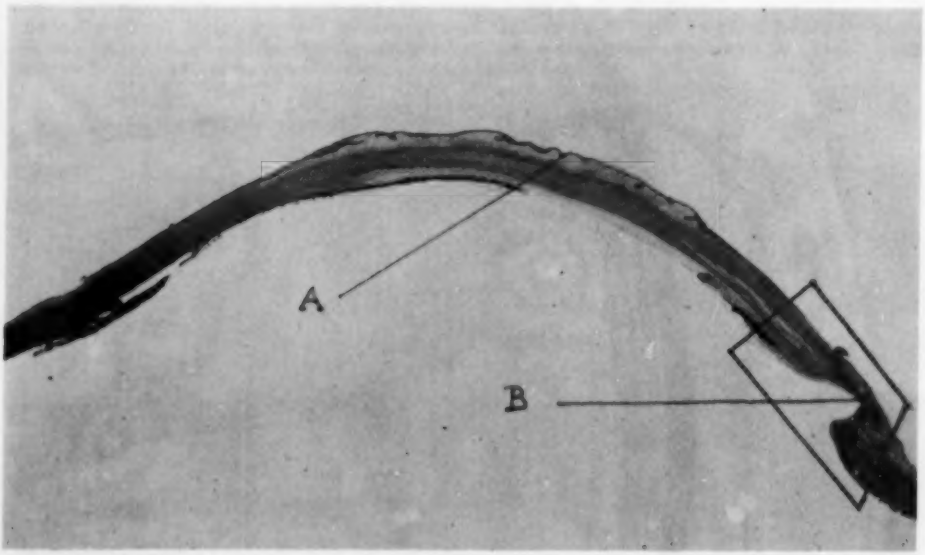


Fig. 14a (Wadsworth). An eye following an intracapsular cataract extraction. The anterior chamber remained flat following operation.

Iris is incarcerated in the wound resulting in poor healing and bulging. The iris is in apposition with the posterior surface of the cornea with an absence of the anterior chamber. With the approximation of the iris to the cornea and the increased intraocular pressure, marked corneal changes have taken place.

There is an irregularity of the epithelium with a fibrous tissue along Bowman's membrane (A). There is an encroachment of the stroma with new-formed vessels. The iris became incarcerated in the wound (B). It was never a strong approximation; therefore, bulging of the wound resulted when the intraocular pressure became high.

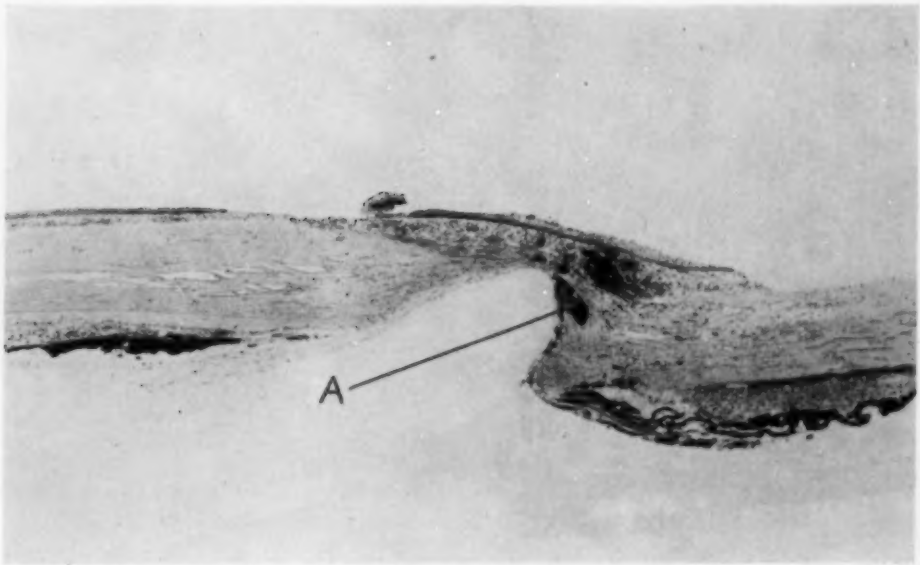


Fig. 14b (Wadsworth). High-power view of area indicated by (B) in Figure 14a. The wound is poorly approximated with iris tissue incarcerated in the scar (A).

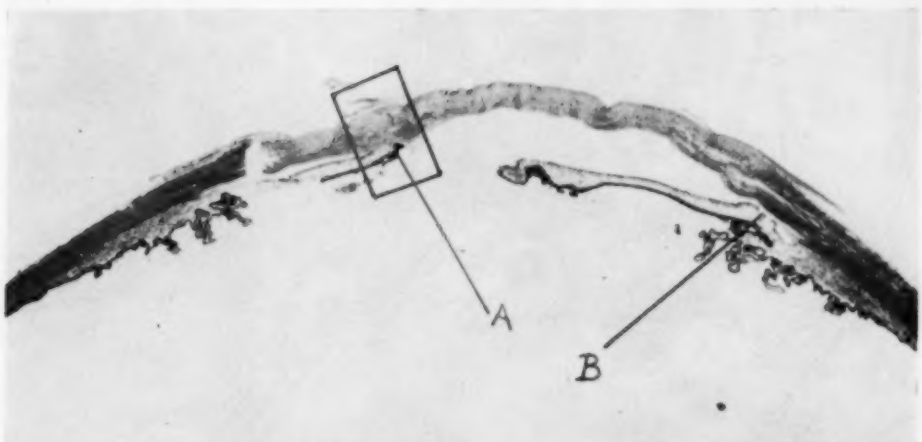


Fig. 15a (Wadsworth). The incision is too far in the cornea. The iris has become adherent to part of the incision (A). There was a delayed restoration of the anterior chamber with fine anterior synechias in the opposite angle (B), but the angle is not completely occluded. Corneal changes are present at the point where the iris is adherent to the cornea.

This eye was enucleated because of discomfort due to recurrent abrasions of the corneal epithelium. The pupil was somewhat updrawn. The anterior chamber was shallow but present.

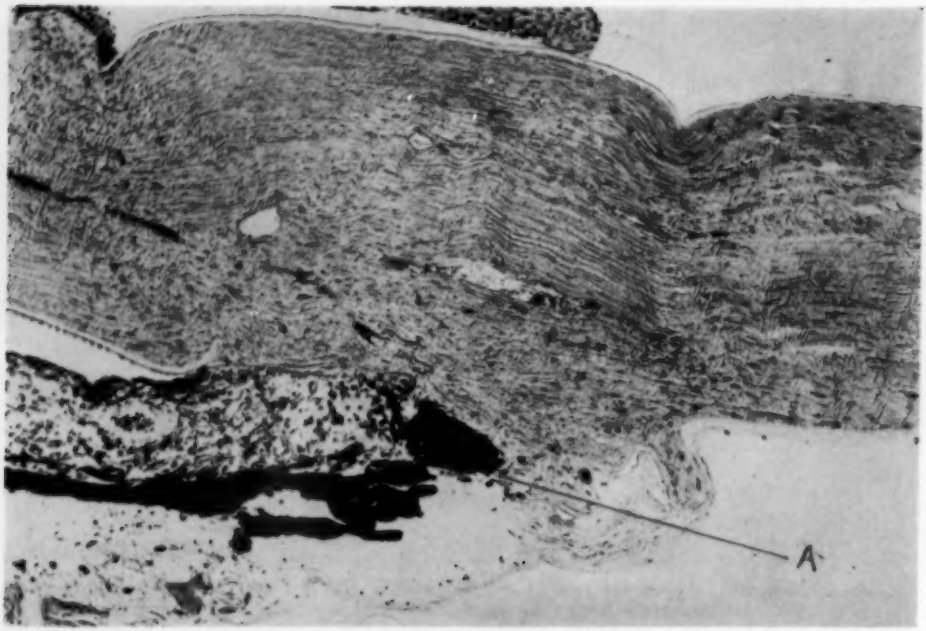


Fig. 15b (Wadsworth). High-power view of area indicated by (A) in Figure 15a. The iris is adherent at the point of incision (A). Descemet's membrane is wrinkled and vascularization of the corneal stroma is present in the region of the incision.

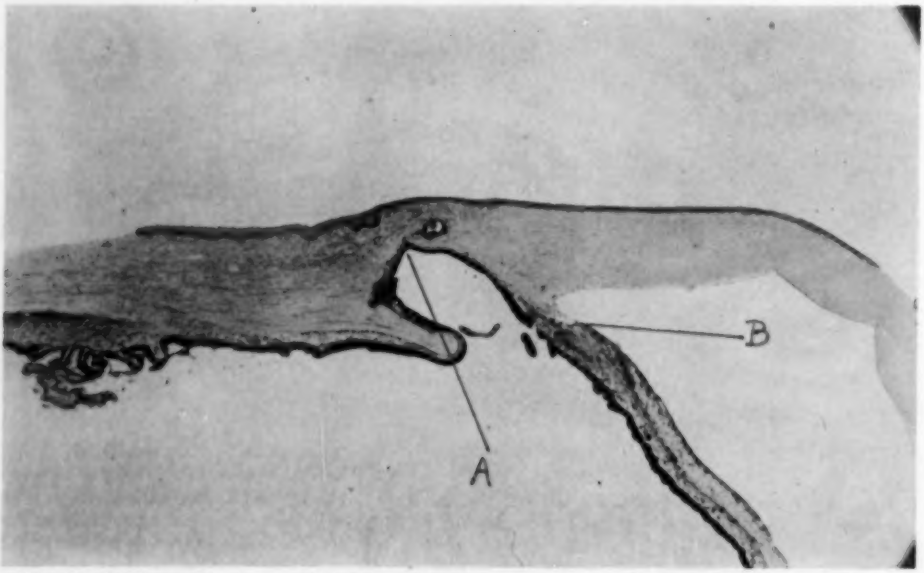


Fig. 16 (Wadsworth). A corneal incision with poor approximation of wound leaving a gap that is lined with iris tissue (A). Loss of vitreous caused updrawn pupil (B).

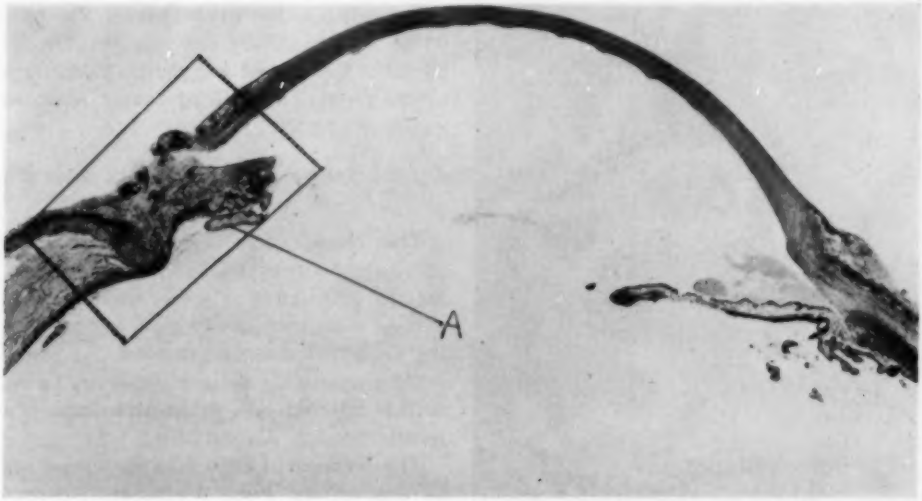


Fig. 17a (Wadsworth). The incision was somewhat scleral with a prolapse of ciliary body into the wound (A).

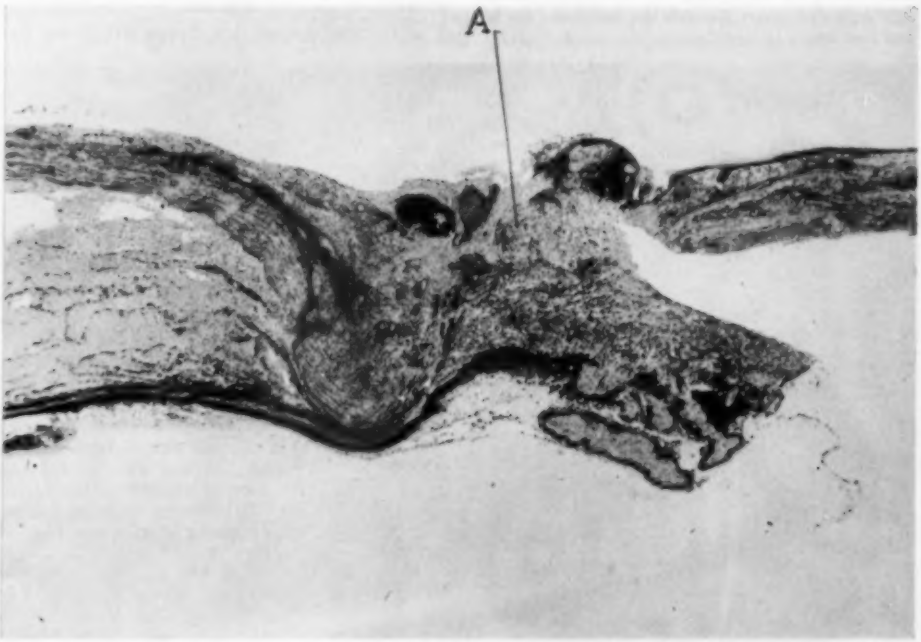


Fig. 17b (Wadsworth). High magnification of area indicated by (A) in Figure 17a. There is almost no attempt at healing and the ciliary body fills the gap between the wound edges (A).

Fig. 18 (Wadsworth). Epithelial downgrowth through the corneal incision following delayed restoration of anterior chamber. There has been poor healing of the wound. The epithelium has not only extended down through the incision (A) but has also begun to line the anterior chamber (B).

Following a low-grade uveitis the posterior surface of the iris may become adherent to the hyaloid and cause an occlusion of the pupil. This will of course result in glaucoma (fig. 21).

Complications following the rupture of the lens capsule

The complications associated with the presence of cortex in the anterior chamber are characterized by a severe uveitis. There may be sufficient cortex remaining to block the angle and cause a glaucoma. As previously mentioned, a definite sensitivity to the cortical material with phacoanaphylactia is a possibility (figs. 22a and 22b).

The presence of capsule in the wound can be very serious. First, it prevents a normal strong healing of the wound. Second, the lens epithelium normally present on the capsule may undergo metaplasia and extend down over the surface of the vitreous and cause grayish lines that can be seen clinically. The contracture of this tissue will draw the

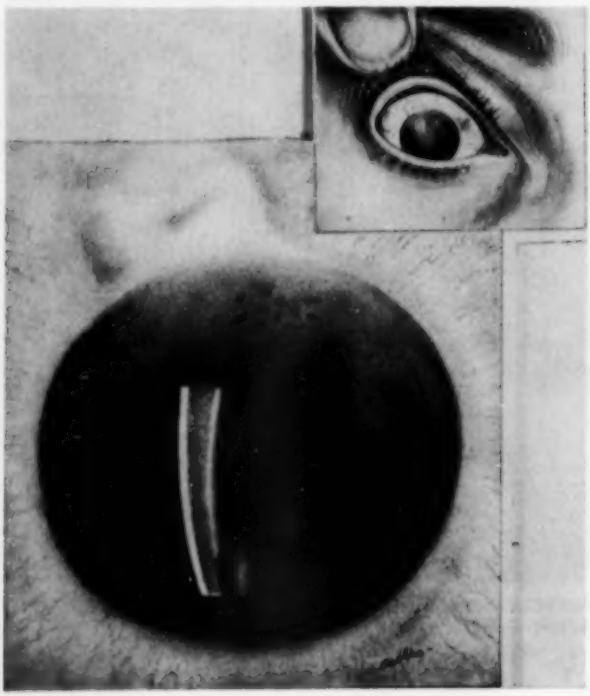


Fig. 19 (Wadsworth). A drawing of epithelial downgrowth that occurred in an eye with a history of delayed restoration of the anterior chamber caused by a leaking wound. The leak was repaired and the anterior chamber promptly reformed. However, epithelization became manifest later.

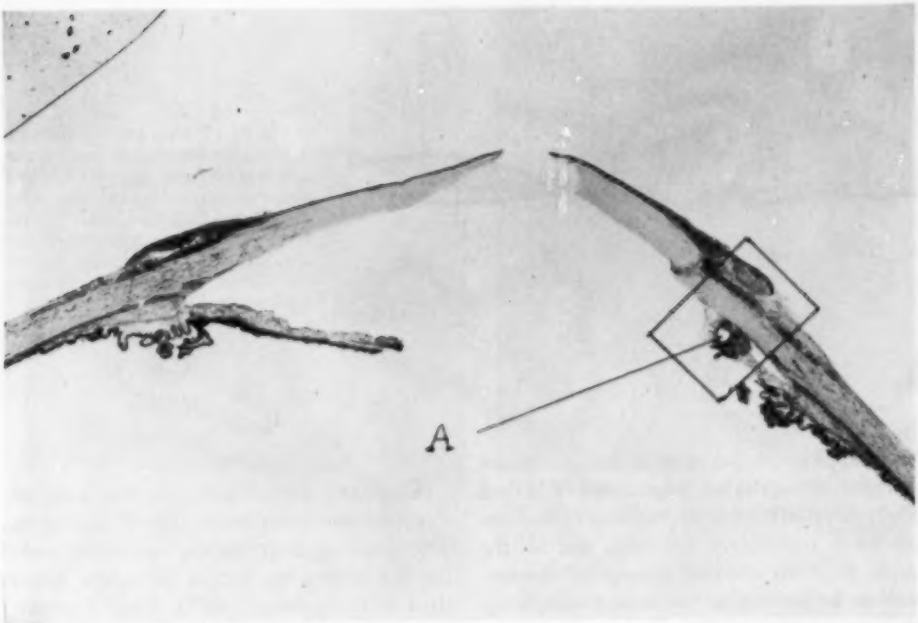


Fig. 20a (Wadsworth). A small solitary epithelial cyst in the iris root (A) following cataract extraction. No further epithelization or downgrowth was observed from multiple sections examined.

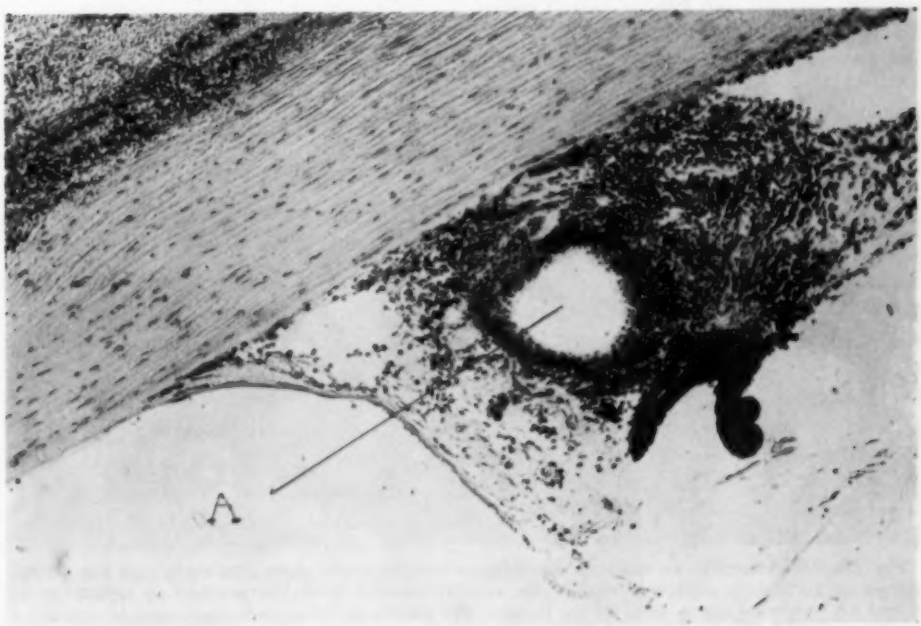


Fig. 20b (Wadsworth). Higher magnification of area indicated by (A) in Figure 20a. The small implantation cyst is self-limited and shows no attempt at lining the anterior chamber (A).

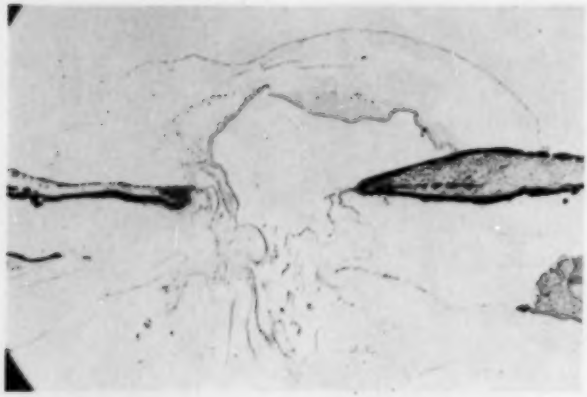


Fig. 21 (Wadsworth). Following a discission the vitreous has become adherent to both the anterior and posterior surface of the iris, completely blocking the pupil. There was a posterior detachment and shrinkage of the vitreous which enabled the vitreous body to move forward with great ease.

pupil up toward the wound and create an eccentric or updrawn pupil (figs. 22a and 22b). A proliferation of the lens epithelium can form translucent spherules that fill the pupil. Such an abortive attempt of the epithelium to form lens fibers may completely obscure vision (figs. 23 and 24).

Hemorrhage

A simple hemorrhage in the anterior chamber may occur at the time of operation. This bleeding is frequently caused by making the section too far in the sclera rather than in the cornea (Lee[12]). Such a hemorrhage is troublesome, but usually does not

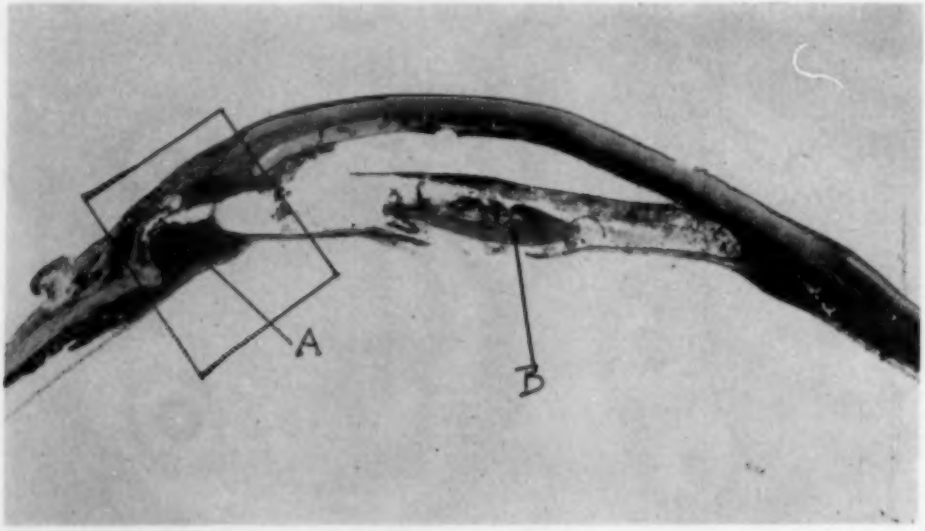


Fig. 22a (Wadsworth). An extracapsular cataract extraction with capsule in the wound and a large amount of cortical material remaining in the anterior chamber (B). The presence of capsule in the wound (A) prevents strong union of the incision. The overwhelming inflammatory response is made up mostly of eosinophils which is highly suggestive of phacoanaphylactia.

Fig. 22b (Wadsworth). High-power view of area indicated in Figure 22a showing capsule (A) and marked inflammatory reaction.

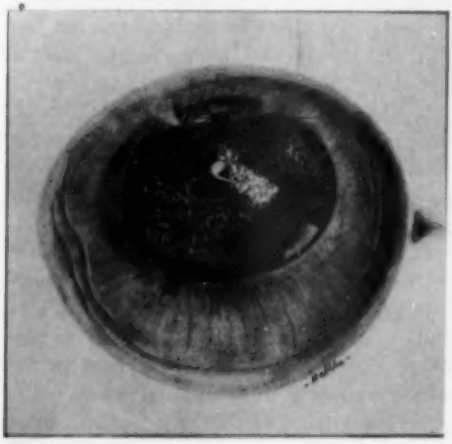


Fig. 23 (Wadsworth). An extracapsular cataract extraction with a gradual loss of vision due to the formation of a secondary membrane caused by the proliferation of the lens epithelium in its attempt to form lens fibers. Here one can see the typical Elschnig bodies filling the pupil.

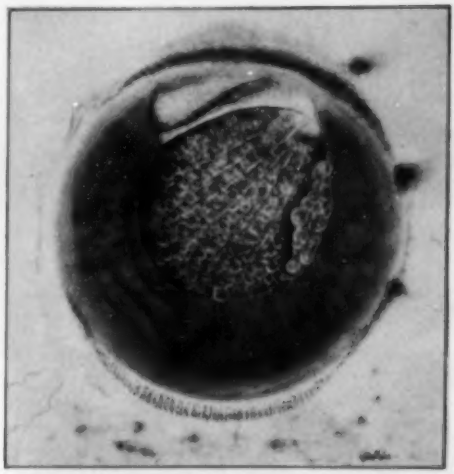


Fig. 24 (Wadsworth). Numerous Elschnig bodies that fill the pupil. Traction lines converge to a point where the capsule is incarcerated in the wound.

result in any complication.

The postoperative hemorrhage that occurs on or about the sixth day is probably the result of a rupture of the small capillaries that have migrated into the scar. At about the sixth day the eye begins to have its normal tension and the scar does not stand the intraocular pressure. With firm corneoscleral sutures these hemorrhages may cause no complications and may absorb within a few days without sequelae.

On the other hand, three things may happen (Owens and Hughes[13]):

1. The hemorrhage may be of sufficient extent to rupture the wound and cause a prolapse of the iris.

2. The blood may enter the vitreous. If the blood extends into the vitreous its absorption is slow and oftentimes incomplete, leaving troublesome vitreous opacities.

3. There may be sufficient hemorrhage to cause glaucoma with multiform complications. Such a condition demands prompt drainage of the blood from the anterior chamber.

The choroidal or expulsive hemorrhage is extremely rare, but if the condition occurs

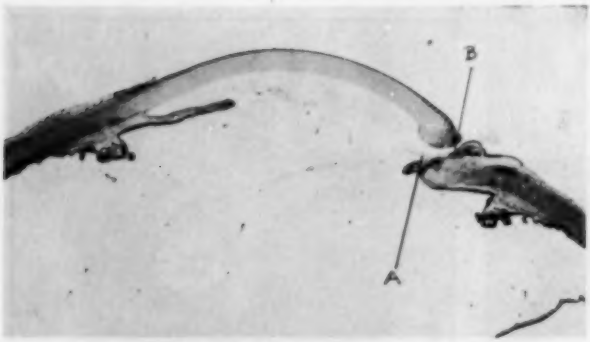


Fig. 25 (Wadsworth). A case in which normal vitreous was lost with incarceration of the vitreous in the wound (A). The wound failed to heal and vitreous remained between the wound edges. A downgrowth of the epithelium along the edges of the wound developed where the vitreous was present between the two edges (B). Cellular elements are beginning to extend down into the formed vitreous and later a contracture of this cellular membrane will cause traction on the retina.

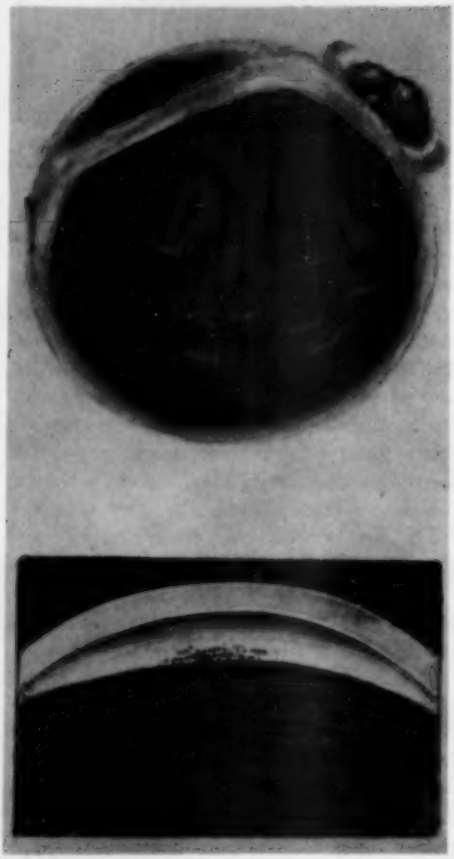


Fig. 26 (Wadsworth). The end result of two cases in which vitreous was lost and iris was incarcerated in the wound. The contracture of the shrinking vitreous caused the iris to be drawn up toward the wound, completely obliterating the pupil.

at the time of operation the eye is often lost. It has been reported that immediate posterior sclerotomy has saved the eye (Verhoeff[14]).

Hemorrhages following retrobulbar injection may cause no complication but simply delay the operation for several days. If the condition is not recognized before the eye is opened, the positive pressure on the globe will cause a loss of vitreous.

Vitreous loss

The role of the vitreous in complications after cataract extraction is varied. There are three types of vitreous and the seriousness of the vitreous loss depends largely on the type of vitreous, and not the amount that is lost.

1. The vitreous may be *completely fluid.* This is most commonly seen in high myopic eyes. A loss of such vitreous is not a very serious situation and may not lead to complications.

2. The so-called *semifluid vitreous,* not the well-formed vitreous that is seen in normal eyes, can lead to formation of a secondary membrane with updrawing of the pupil. The incarceration of vitreous in the wound causes a weak approximation.

3. The loss of *normal vitreous jell* causes serious complications. It can shrink and cause the iris to be drawn up toward the wound. If there is an adhesion of the vitreous to the retina a detachment of the retina may develop. The eye will remain

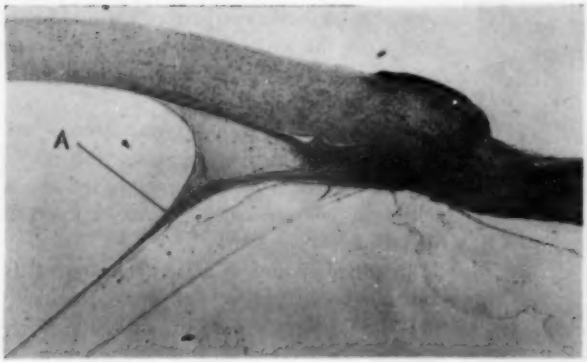


Fig. 27 (Wadsworth). The vitreous is adherent to the wound (A) with a dense cellular membrane extending to the retina. The contracture of this membrane has caused a detachment. Such a detachment of course would be quite unfavorable to the usual diathermy procedure.

irritable for many weeks following the operation and a secondary glaucoma often results due to the distortion of the anterior chamber from the contracture of the vitreous or from a secondary inflammatory reaction that often occurs with the loss of normal vitreous (Kirby[15]) (figs. 25 and 26).

Detachment of the retina

A detachment of the retina may present

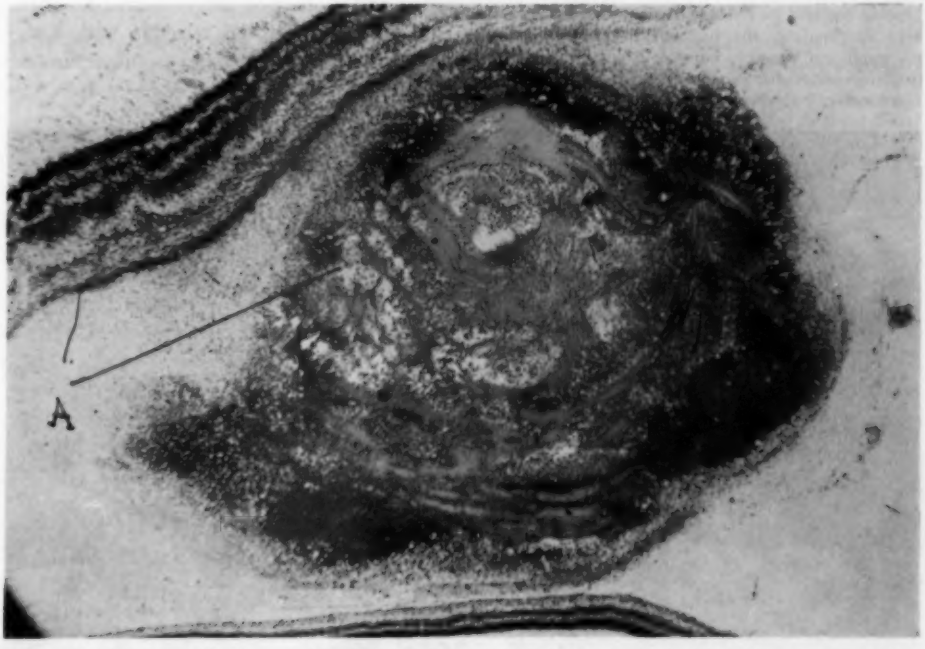


Fig. 28 (Wadsworth). An eye in which the lens was inadvertently pushed into the vitreous cavity. Apparently the capsule was removed but the nucleus remained in the vitreous (A).

A severe reaction resulted and the shrinkage of the vitreous caused a complete detachment of the retina and choroid as well. In this case the inflammatory reaction came on rapidly after the lens was lost.

In cases of couching, the lens is covered by capsule and the toxic products do not escape into the vitreous so rapidly.

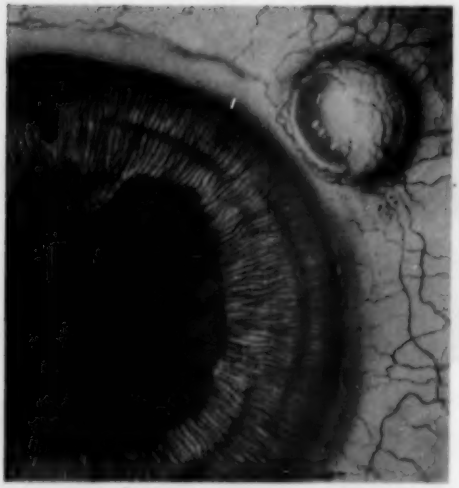


Fig. 29 (Wadsworth). Proliferation of pigment on the posterior surface of the cornea. This small pigment area grew in size over a period of a year. During the last two years, it has remained stationary. Fortunately, the pigment does not obstruct the pupil and vision is normal. Along the conjunctival incision there is a small epithelial cyst. This spontaneously disappeared.

itself in an aphakic eye just as it would in an eye before the lens has been removed. Such cases have a definite hole and their prognosis is good for reattachment.

If, however, there has been vitreous loss in an eye with normal vitreous, the retina may be pulled from its attachments and a diathermy operation does not often reattach the retina (Townes[16]).

An inflammation in the vitreous with its subsequent shrinkage may cause a detachment of the retina. In such cases the prognosis is very poor because the vitreous is organized with dense strands and mechanically separates the retina. In such a case a diathermy operation cannot pull the retina back to its normal position (fig. 27).

If the lens is lost in the vitreous and remains there, a marked inflammatory reaction will result. Following this inflammation there is a shrinkage of the vitreous with a detachment of the retina and complete loss of vision (fig. 28).

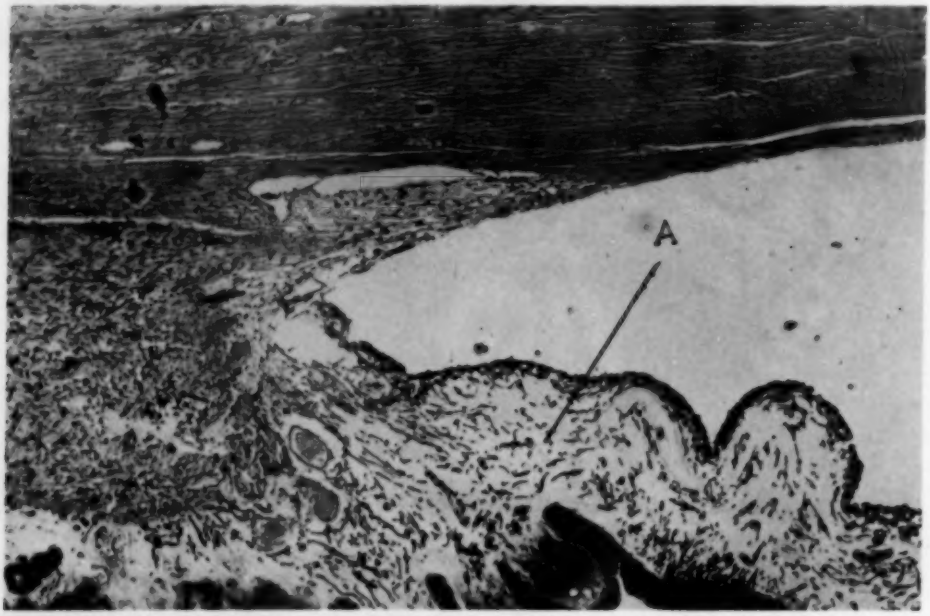


Fig. 30 (Wadsworth). This section shows the base of an iris in a normal eye (A). There is considerable stromal tissue at the base of the iris and one would expect this to be relatively strong.

Fig. 31 (Wadsworth). The base of the iris (A) is thinner than that of the previous section. Here, undue traction would cause the iris to tear more easily than in the preceding case. This condition is amplified in elderly people in whom atrophy further weakens the iris tissue.

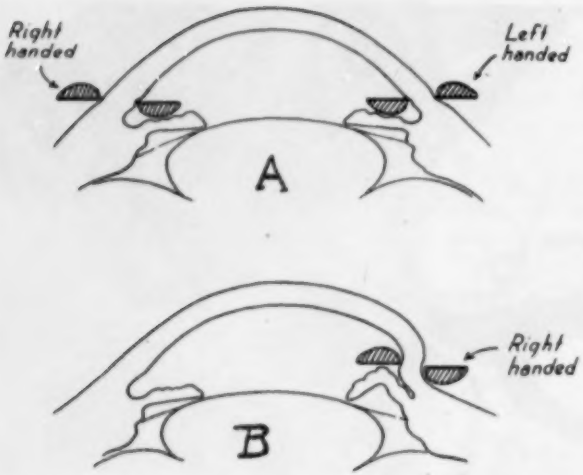
As was described by Atkinson,[17] a proliferation of pigment on the posterior surface of the cornea may follow cataract extraction. If this pigment is centrally placed, it will markedly interfere with vision (fig. 29).

On occasions, an iridodialysis is produced during the enlargement of the incision with scissors. There may be several reasons for the production of such an iridodialysis:

First, there is considerable normal variation in the thickness of the iris at its base. Traction exerted on the iris will cause it to tear at the base and produce an iridodialysis. If the iris is unusually thin at its base, the slightest traction may produce an iridodialysis (figs. 30, 31, and 32).

The use of right-handed and left-handed scissors while enlarging the section may help to prevent such a complication (fig. 33). The effect of right-handed scissors along the right side of the incision causes buckling of the cornea and distortion of the scleral ring. Under such circumstances, the iris is knuckled into the opening of the scissors and an iridotomy is formed. Also, if there is a weakened area at the base of the iris there may be sufficient traction to produce an iridodialysis.

Fig. 32 (Wadsworth). This shows a large iridodialysis produced while enlarging the corneal section. The postoperative course was uneventful but the patient developed monocular diplopia. The iridodialysis was corrected by using a blunt Turell hook and pulling the iris into a small incision at the limbus and touching this hook with diathermy. There was sufficient adhesion at this point to cause the iris to adhere there. The pupil then became round and the diplopia disappeared (Goar and Schultz[20]).



SCISSOR SECTION IN CATARACT EXTRACTION

Fig. 33 (Wadsworth). A drawing in which the right-handed scissors are used on the left side of the incision and the left-handed scissors are used on the right side (A). One can see that the line of cutting is perpendicular to the cornea. This causes a minimum distortion of the globe during the use of the scissors. When right-handed scissors are used on the right side of the incision, a buckling of the cornea results (B).

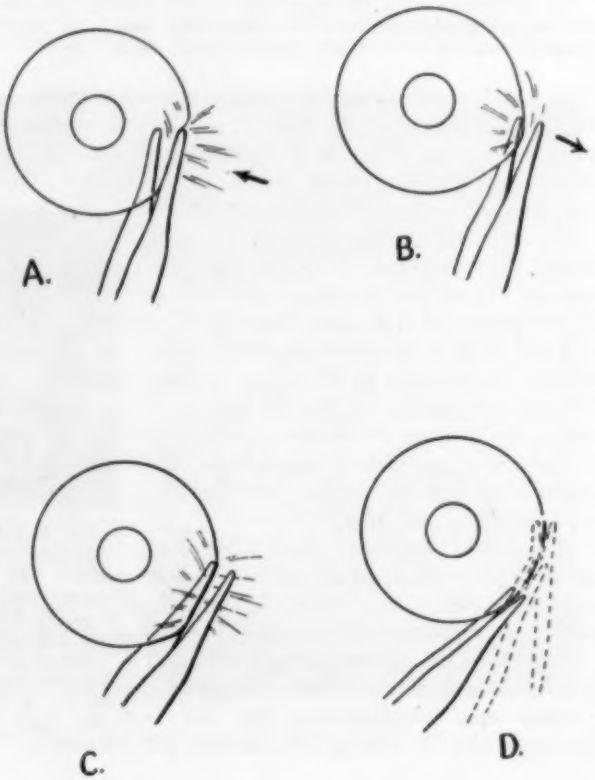


Fig. 34 (Wadsworth). This is a drawing which shows some of the common errors in the use of the scissors:

(A) If, in closing the scissors, there is more force exerted by the blade that is outside the anterior chamber, distortion of the cornea and scleral ring will result.

(B) If the opposite is true, distortion in the opposite direction will result.

(C) If too large a bite is taken in the scissors, this tends to straighten the limbal ring and the iris may be swept into the scissors. The scleral ring may be distorted as well.

(D) The last drawing shows the more ideal method for using scissors. That is, to take small bites and to have the scissors parallel with the curvature of the cornea, changing one's position with each snip of the scissors.

When the incision is enlarged with scissors, every effort should be made to cause the least amount of distortion to the limbal ring.

If, in making the bite with the scissors, a greater amount of pressure is exerted on the blade outside of the eye, the pressure on the globe may straighten the limbal curve sufficiently to cause a rupture of the zonules.

If the opposite is true and a greater force is exerted on the blade inside the eye, a distortion of the cornea and limbal ring is sufficient to cause an iridodialysis.

Too large a bite with the scissors will also flatten the scleral ring and cause the iris to be swept beneath the blade of the scissors resulting either in an iridodialysis or a large iridotomy (fig. 34-A, B, C).

If the scissors are closed with equal pressure on both sides of the cornea, minimal distortion will result. It is advisable to make multiple small bites and attempt to have the scissors parallel with the portion of the limbus being cut. In so doing, it will be necessary to change the position of the scissors with each bite following the curve of the limbus (fig. 34-D).

Summary

Many common complications following cataract extraction have been presented. When possible, the conditions have been documented with microscopic sections.

By knowing the cause of these complications it is often possible to prevent a great majority of the troublesome sequelae resulting from cataract surgery.

The value of left-handed scissors was emphasized.

73 East 71st Street (21).

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A COMPARISON OF REFRACTION RESULTS ON THE SAME INDIVIDUALS*

ALBERT E. SLOANE,† M.D., EDWIN B. DUNPHY,‡ M.D., WILLIAM V. EMMONS,§ M.D., AND J. ROSWELL GALLAGHER,‖ M.D.
Boston, Massachusetts

The reliability of various screening devices has been reported by Imus[1] and, in the discussion of his paper, others have commented upon the variability of refraction results when the same patient is examined by different specialists or even by the same specialist at different times. An opportunity to observe the extent of variation in refraction findings has recently been provided us in connection with a study of the effect of a training method upon myopia.[2]

As part of that study two ophthalmologists and one optometrist examined, on the same day and under similar conditions, the eyes of 21 different adolescents. The subjects were far above average intelligence, the conditions of the examination ideal, the tests were made in an efficient but leisurely manner, and the examiners made their best efforts to achieve accuracy. Although the number of subjects examined is small, it would seem that these data offer valid evidence in regard to the consistency of clinical findings.

METHOD

The 21 subjects ranged from 14 to 18 years of age. A cycloplegic was not used. Each subject was tested by each of the examiners within a two-hour period of the same day. Each examiner used the method of refraction he was accustomed to use in his office practice. Tests for heterophoria (Maddox rod) were made on only 10 boys and these tests were not repeated. All the results were obtained and were recorded independently, and the examiners did not compare their findings with each other. The findings were turned over to one of us (J. R. G.) for future reference and analysis.

RESULTS AND COMMENT

Table 1 lists the naked vision findings obtained by each of the three examiners on each of the 21 subjects.

No attempt has been made to present a quantitative evaluation of these data. Efforts to apply statistical methods proved unsatisfactory and, after considerable thought, it was decided that all such attempts would yield misleading results. Translation of the findings into spheric equivalents was considered as a possible means of comparing one examiner's findings with another but this was discarded because the presence of multiple variables such as the axis of astigmatism precluded the use of such a method. It has seemed to us best only to present the actual findings and to invite an inspection of Table 1.

On no one subject did all three examiners obtain identical naked vision, and to only one (T. S.) did they give the same correction. The differences between their findings, however, are not great and are probably well within each subject's limit of tolerance. It seems clear that test findings will differ from examiner to examiner even under the best conditions and that one cannot properly be dogmatic about a given refractive error. The variations shown in Table 1 may reasonably be considered to be of the order one can expect under ideal conditions.

* From the Massachusetts Eye and Ear Infirmary and the Adolescent Unit, Children's Medical Center, Boston. This study was supported by funds from The Grant Foundation, Inc.

† Director, Department of Refraction, Massachusetts Eye and Ear Infirmary.
‡ Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary.
§ Department of Refraction, Massachusetts Eye and Ear Infirmary.
‖ Chief of the Adolescent Unit, Children's Medical Center.

TABLE 1

NAKED VISION, CORRECTION, AND CORRECTED VISION FINDINGS OBTAINED BY THREE EXAMINERS WHEN 21 SUBJECTS WERE TESTED BY EACH OF THEM ON THE SAME DAY

Subject	Examiner	Naked Vision		Correction	Corrected Vision
L. B.	AES	OD	12/200	−4.0D. sph. ◯ −0.25D. cyl. ax. 180°	20/15
		OS	10/200	−4.5D. sph. ◯ −1.0D. cyl. ax. 180°	20/25
	EBD	OD	20/200	−4.0D. sph. ◯ −0.5D. cyl. ax. 180°	20/15
		OS	20/200	−4.25D. sph. ◯ −1.0D. cyl. ax. 180°	20/15
	WVE	OD	10/200	−4.25D. sph.	
		OS	10/200	−4.5D. sph. ◯ −0.5D. cyl. ax. 180°	
J. B.	AES	OD	15/200	−4.25D. sph.	20/20
		OS	15/200	−4.5D. sph. −0.25D. cyl. ax. 105°	20/20
	EBD	OD	15/200	−4.25D. sph. ◯ −0.12D. cyl. ax. 90°	20/15
		OS	10/200	−4.37D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
	WVE	OD	20/200	−4.5D. sph.	
		OS	20/200	−4.5D. sph.	
P. C.	AES	OD	20/40–20/30	−0.62D. sph.	20/15
		OS	20/15	+0.5D. sph.	20/10
	EBD	OD	20/70	−0.5D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
		OS	20/15	+0.75D. sph. ◯ −0.25D. cyl. ax. 85°	20/15
	WVE	OD	$20/40^{-1}$	−0.5D. sph.	
		OS	20/15	+0.5D. sph.	
J. D.	AES	OD	20/100	−1.5D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
		OS	20/200	−3.25D. sph. ◯ −0.25D. cyl. ax. 90°	20/20
	EBD	OD	20/40	−1.5D. sph. ◯ −0.5D. cyl. ax. 90°	20/15
		OS	20/200	−3.0D. sph. ◯ −0.5D. cyl. ax. 90°	20/15
	WVE	OD	$20/40^{-1}$	−2.25D. sph.	
		OS	20/200	−3.5D. sph.	
H. H.	AES	OD	20/200	−3.0D. sph. ◯ −0.5D. cyl. ax. 180°	20/30
		OS	20/100	−2.0D. sph. ◯ −0.5D. cyl. ax. 180°	20/15
	EBD	OD	20/100	−2.75D. sph. ◯ −0.25D. cyl. ax. 175°	20/15
		OS	20/70	−2.25D. sph. ◯ −0.25D. cyl. ax. 5°	20/15
	WVE	OD	20/200	−3.25D. sph.	
		OS	20/80	−2.75D. sph.	
E. H.	AES	OD	15/200	−3.75D. sph. ◯ −0.25D. cyl. ax. 165°	20/15
		OS	15/200	−2.75D. sph. ◯ −0.75D. cyl. ax. 15°	20/15
	EBD	OD	9/200	−3.75D. sph.	20/15
		OS	12/200	−2.5D. sph. ◯ −0.75D. cyl. ax. 155°	20/15
	WVE	OD	9/200	−4.0D. sph.	
		OS	10/200	−2.75D. sph. ◯ −0.75D. cyl. ax. 165°	
A. J.	AES	OD	20/50	−0.75D. sph. ◯ −0.5D. cyl. ax. 90°	20/15
		OS	20/50	−0.75D. sph. ◯ −0.25D. cyl. ax. 60°	20/15
	EBD	OD	20/50	−0.25D. sph. ◯ −0.5D. cyl. ax. 105°	20/15
		OS	20/50	−0.5D. sph. ◯ −0.5D. cyl. ax. 75°	20/15
	WVE	OD	$20/40^{-1}$	−0.75D. sph. ◯ −0.75D. cyl. ax. 90°	
		OS	$20/50^{+1}$	−0.75D. sph. ◯ −0.75D. cyl. ax. 90°	
R. S.	AES	OD	20/70	−0.75D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
		OS	20/200	−1.25D. sph. ◯ −0.5D. cyl. ax. 90°	20/15
	EBD	OD	20/60	−1.0D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
		OS	20/80	−1.25D. sph. ◯ −0.25D. cyl. ax. 90°	20/15
	WVE	OD	$20/70^{-1}$	−0.75D. sph.	
		OS	$20/80^{-1}$	−1.75D. sph.	
T. S.	AES	OD	20/15	plano	20/15
		OS	20/100	−1.75D. sph.	20/15
	EBD	OD	20/15	plano	20/15
		OS	20/100	−1.75D. sph.	20/15
	WVE	OD	20/20	plano	
		OS	20/200	−1.75D. sph.	
W. S.	AES	OD	12/200	−2.25D. sph. ◯ −0.75D. cyl. ax. 135°	20/15
		OS	12/200	−2.25D. sph. ◯ −0.5D. cyl. ax. 75°	20/15
	EBD	OD	18/200	−2.0D. sph. ◯ −0.62D. cyl. ax. 135°	20/15
		OS	19/200	−2.0D. sph. ◯ −0.5D. cyl. ax. 75°	20/15
	WVE	OD	20/200 dim	−2.25D. sph. ◯ −0.5D. cyl. ax.135°	
		OS	20/200 dim	−2.0D. sph. ◯ −0.75D. cyl. ax. 60°	
R. T.	AES	OD	12/200	−3.25D. sph.	20/15
		OS	20/200	−2.75D. sph. ◯ −0.5D. cyl. ax. 180°	20/15
	EBD	OD	12/200	−3.5D. sph. ◯ −0.12D. cyl. ax. 25°	20/15
		OS	20/100	−3.0D. sph. ◯ −0.25D. cyl. ax. 15°	20/15

TABLE 1—(*Continued*)

Subject	Examiner		Naked Vision	Correction	Corrected Vision
	WVE	OD	20/80^{-1}	−3.5D. sph.	
		OS	20/80^{-2}	−2.75D. sph.◯−0.5D. cyl. ax. 180°	
W. Y.	AES	OD	20/200^{+1}	−2.0D. sph.◯−0.25D. cyl. ax. 90°	20/20
		OS	20/200^{+1}	−2.25D. sph.	20/15
	EBD	OD	20/80	−2.25D. sph.◯−0.25D. cyl. ax. 90°	20/15
		OS	20/100	−2.25D. sph.	20/15
	WVE	OD	20/200	−2.25D. sph.◯−0.75D. cyl. ax. 90°	
		OS	20/200	−2.25D. sph.	
D. B.	AES	OD	20/50	−0.62D. sph.	20/15
		OS	20/50^{+2}	−0.5D. sph.◯−0.25D. cyl. ax. 180°	20/15
	EBD	OD	20/70^{+}	−1.0D. sph.	20/15
		OS	20/70^{+}	−0.62D. sph.◯−0.75D. cyl. ax. 180°	20/15
	WVE	OD	20/70	−0.75D. sph.	
		OS	20/70	−0.75D. sph.	
J. C.	AES	OD	20/200	−2.75D. sph.◯−0.5D. cyl. ax. 90°	20/15
		OS	20/100	−2.75D. sph.◯−0.5D. cyl. ax. 10°	20/15
	EBD	OD	20/100	−3.12D. sph.	20/20^{+}
		OS	20/100	−3.0D. sph.◯−0.25D. cyl. ax. 85°	20/15
	WVE	OD	20/100	−3.5D. sph.	
		OS	20/100	−3.5D. sph.	
W. D.	AES	OD	20/40	−0.62D. sph.	20/15
		OS	20/40	−0.5D. sph.◯−0.25D. cyl. ax. 90°	20/15
	EBD	OD	20/50	−0.75D. sph.	20/15
		OS	20/50	−0.75D. sph.	20/15
	WVE	OD	20/70	−0.5D. sph.◯−0.25D. cyl. ax. 180°	
		OS	20/70	−0.5D. sph.	
L. L.	AES	OD	20/200	−2.25D. sph.◯−0.25D. cyl. ax. 90°	20/15
		OS	20/100	−1.5D. sph.◯−0.5D. cyl. ax. 105°	20/15
	EBD	OD	20/100	−1.75D. sph.◯−0.25D. cyl. ax. 95°	20/15
		OS	20/100 ±	−1.5D. sph.◯−0.25D. cyl. ax. 85°	20/15
	WVE	OD	20/200	−2.5D. sph.	
		OS	20/200	−2.5D. sph.	
R. R.	AES	OD	20/70	−1.25D. sph.◯−0.25D. cyl. ax. 90°	20/15
		OS	20/30	+0.25D. sph◯−0.5D. cyl. ax. 120°	20/30
	EBD	OD	20/70	−1.5D. sph.	20/15
		OS	20/40	−0.5D. sph.	20/25
	WVE	OD	20/70^{+1}	−1.5D. sph.	
		OS	20/30	−0.5D. sph.	
J. S.	AES	OD	20/50	−1.75D. sph.◯−0.25D. cyl. ax. 90°	20/15
		OS	20/20	+0.5D. sph.◯−0.25D. cyl. ax. 90°	20/15
	EBD	OD	20/70	−1.75D. sp .◯−0.25D. cyl. ax. 95°	20/15
		OS	20/15	+0.62D. sph.	20/15
	WVE	OD	20/100	−1.75D. sph.	
		OS	20/20^{+3}	+0.25D. sph.	
K. S.	AES	OD	20/200	−2.5D. sph.◯−0.5D. cyl. ax. 75°	20/15
		OS	20/200^{+1}	−3.25D. sph.◯−0.5D. cyl. ax. 105°	20/15
	EBD	OD	20/200	−2.75D. sph.◯−0.5D. cyl. ax. 65°	20/15
		OS	15/20^{-}	−3.5D. sph.◯−0.5D. cyl. ax. 95°	20/15
	WVE	OD	20/200	−3.25D. sph.	
		OS	20/200	−3.75D. sph.◯−0.5D. cyl. ax. 90°	
R. S.	AES	OD	15/200	−3.75D. sph.	20/15
		OS	13/200	−3.75D. sph.	20/15
	EBD	OD	18/200	−3.5D. sph.◯−0.5D. cyl. ax. 20°	20/15
		OS	15/200	−3.75D. sph.◯−0.25D. cyl. ax. 165°	20/15
	WVE	OD	5/50^{-2}	−4.0D. sph.◯−0.5D. cyl. ax. 45°	
		OS	5/50^{-3}	−4.5D. sph. (Add +0.5D. sph., O.U.)	
R. W.	AES	OD	20/60	−0.62D. sph.◯−0.25D. cyl. ax. 90°	20/15
		OS	20/20	plano	20/15
	EBD	OD	20/40^{-}	−0.75D. sph.◯−0.25D. cyl. ax. 120°	20/15
		OS	20/20	−0.25D. sph.	20/15
	WVE	OD	20/70	−0.5D. sph.	
		OS	20/20	plano	

TABLE 2

MEASUREMENTS OF HETEROPHORIA FOR DISTANCE AND NEAR BY EACH OF THE THREE EXAMINERS

Subject	AES			EBD			WVE		
	Vertical Distant	Horizontal		Vertical Distant	Horizontal		Vertical Distant	Horizontal	
		Distant	Near		Distant	Near		Distant	Near
D. B.	Ortho	Ortho	1 Exo	Ortho	1 Eso	2 Eso	Ortho	2 Eso	1 Eso
J. C.	½ L.H.	4 Exo	?	1 L.H.	2 Exo	1 Eso	1 (?)	5 Exo	1 Eso
W. D.	Ortho	1 Exo	2 Exo	½ L.H.	1 Exo	4 Exo	Ortho	½ Exo	4 Exo
L. L.	Ortho	10 Eso	4 Eso	1 L.H.	4 Eso	6 Eso	Ortho	7 Eso	6–7 Eso
R. R.	(Alternating Exotropia)				—			—	
J. S.	Ortho	Ortho	Ortho	Ortho	3 Exo	Ortho	Ortho	3 Exo	Ortho
K. S.	Ortho	1 Eso	1 Eso	1 R.H.	Ortho	1 Eso	2½ (?)	Ortho	2–3 Eso
W. S.	Ortho	Ortho	4 Eso	1 L.H.	2 Exo	Ortho	Ortho	1 Exo	Ortho
R. S.	Ortho	Ortho	Ortho	Ortho	6 Exo	6 Exo	Ortho	6 Exo	1 Exo
R. W.	Ortho	2 Eso	4 Eso	Ortho	2 Eso	3 Eso	Ortho	1 Eso	4–5 Eso

Table 2 lists the measurements of heterophoria for distance and near. No attempt has been made to evaluate the reason for the variation in findings.

SUMMARY

1. Three examiners tested the vision of 21 adolescents on the same day and under the same conditions. The naked vision, correction, and corrected vision recorded by each examiner are tabulated.

2. In only one instance were the findings of the three examiners identical. The variations, however, were not great and were considered to be well within each subject's tolerance.

3. These data indicate that differences in test findings are inevitable even under the best conditions; that differences of the degree shown in these data may be considered "expected variations"; and that one cannot reasonably be dogmatic about a refractive error.

243 Charles Street (14).

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A PSYCHOMETRIC EVALUATION OF ORTHO-RATER AND WALL-CHART TESTS*

D. A. GORDON, J. ZEIDNER, H. J. ZAGORSKI, AND J. E. UHLANER
Washington, D.C.

Although visual acuity tests are customarily given on wall charts, other methods of presentation have been suggested from time to time. Slides of acuity symbols have been projected on screens, with provision for enlarging and reducing the image.[7] Instruments have been devised which permit mechanical change in size,[10] distance,[5] and orientation[4] of the test object. None of these innovations has achieved as wide an acceptance as wall-chart testing.

Recently, several instruments involving

* From the Personnel Research Branch, TAGO, Department of the Army. Any opinions expressed herein are those of the authors and do not necessarily reflect those of the Department of the Army.

optical simulation of distance have been developed for large scale acuity testing. Among such devices are the Bausch and Lomb Ortho-Rater, the Keystone Telebinocular, and the American Optical Company Sight-Screener. These instruments provide a means of presenting tests of right eye, left eye, and binocular acuity, as well as vertical and horizontal phoris, stereopsis, and color vision. Both near and far simulated distances may be used.

For the measurement of far visual acuity, these instruments have several important advantages over wall-chart testing. The light source is built in and, therefore, can be made relatively accurate. Alley charts, on the contrary, vary widely in condition of illumination. The viewing distance of instruments is achieved optically, with consequent economy of testing space. Targets may be conveniently changed without crossing the testing room. On the same instrument, a number of visual functions may be tested.

Since instrument tests involve a somewhat different optical principle than those of wall charts, a comparison should be made between results on the two methods of presentation. Before instrument tests can be accepted, they must be shown to have satisfactory reliability; that is consistency in repeated measurement.* It must also be demonstrated that the instrument test measures essentially the same visual abilities as the wall-chart test. The present paper deals with these problems.

The reliability of wall-chart tests of far visual acuity has been determined.[2] Data are also available on the reliability of instrument tests.[1,3] A rigorous comparison between these reliabilities cannot be made because of differences in the population, test targets, and light levels employed.

A study by Sulzman, Cook, and Bartlett[9] did employ the same subjects in comparing the reliabilities of instrument and of wall-chart tests. The instruments they employed included the Sight-Screener, the Ortho-Rater, and the Telebinocular.

It was found that the reliabilities of the letter wall-chart tests were about the same as those of the instrument tests. They ranged from 0.80 to 0.88 for the two wall-chart tests, and between 0.81 and 0.85 for the three instrument tests. In near visual acuity testing, reliabilities were also similar. The wall charts, however, seemed to be testing a visual function somewhat different from that of the instrument tests. The correlation between the letter wall-chart tests was considerably higher than those between wall and instrument tests. If these correlations had been corrected for attenuation, the differences would be even larger.

The authors conclude that these results may be due to the introduction of some new factor related to the optical system of the instrument or to the fact that different targets are used in the various tests.

Altman and Rowland[3] determined the relationship between scores obtained on an Ortho-Rater and a wall chart when the same target was used. The wall chart was an accurate enlargement of the plate reproduced for presentation at 20 feet. One hundred and 57 eyes were tested without refractive corrections in order to secure a wide range of acuity scores. A correlation of 0.94 was obtained between acuity scores on the Ortho-Rater and wall-chart tests. This study presents convincing evidence of the identity of the visual abilities measured by the two methods.

In the present experiment, an attempt was made to compare the test-retest reliabilities and to obtain a measure of the correspondence between scores on the Ortho-Rater and wall-chart tests. The same subjects, targets,

* Reliability is measured by the correlation between successive measures on the same instrument. Correlations vary between +1.00 and −1.00. A correlation of +1.00 indicates perfect correspondence between measures. A correlation of −1.00 indicates perfect inverse relationship between scores. A correlation of 0 indicates a completely random relationship between measures.

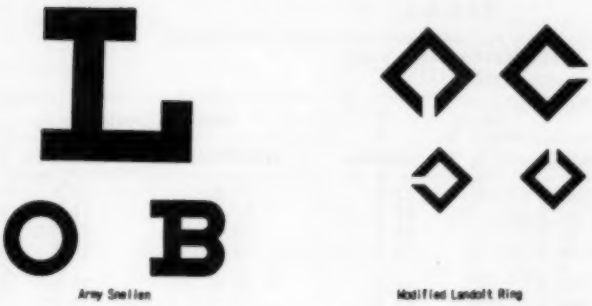

Fig. 1 (Gordon, et al) Visual acuity tests.

and light level were employed in both methods of presentation. The conditions of luminance and contrast between object and background were equalized as closely as possible. With control of these conditions, more definite conclusions may perhaps be reached concerning reliability of the two presentation methods, and the presence or absence of the "apparatus accommodation" factor thought by some to affect machine scores.[5]

Method and Procedure

The present experiment was conducted at the Personnel Research Branch's Pentagon Laboratory in Washington, D.C. The subjects were 117 soldiers from Fort Myer, Virginia. Soldiers varied in age between 19 and 37 years, with the mean age at 22.4 years. The test targets were observed binocularly. All subjects who customarily wore corrective lenses used them in the experiment.

The test designs included a letter chart and a modified Landolt ring chart.* Samples of the items in these targets are shown in Figure 1. The letter chart was a modification of the Snellen Chart employed by the Army in routine visual acuity examination. The chart consisted of 12 lines of letters ranging in size from 20/100 to 20/7.1 Snellen. The modified Landolt ring chart presented a square target rather than the circular design used in the original ring. The chart contained 11 lines of items, ranging in size from 20/135.2 to 20/5.9 Snellen.

The Ortho-Rater plates were made from the wall charts by a double reduction photographic process. It was intended to reduce the wall charts (constructed for testing at 20 feet) to 0.0555 of the original size.† Actually, the reduction ratios, as determined by an optical comparator with microscopic attachment, are: letter plate, right eye 0.0546, left eye 0.0545; Landolt plate, right eye 0.0552, left eye 0.0549. The visual angles corresponding to the reduction ratios of the Ortho-Rater letter targets are slightly smaller than those of the counterpart wall chart; the visual angles of the Ortho-Rater Landolt targets are almost identical to their wall chart.

The laboratory in which testing took place was constructed in conformity to specifications formulated by the Armed Forces—NRC Vision Committee. The viewing distance was 20 feet for wall-chart testing. Illumination was furnished by three overhead lights in flashed opal glass fixtures. These fixtures were evenly spaced along the testing alley. The front of the alley, sides, top, and floor were covered by white osnaburg cloth which served to provide an evenly lit surround over the visual field.

The brightness of the wall charts and

* We wish to acknowledge our indebtedness to Mr. Owen Conger, Typo and Design Unit, Army Publications, Service Branch, TAGO, for his careful drafting of these vision targets.

† This reduction ratio is employed by the Bausch and Lomb Company in the manufacture of Ortho-Rater plates. It is based on an estimated distance of 40 mm. from lens surface to the eye, and 362 mm. from the far slide to the eye.

TABLE 1

COMPARISON OF MEAN AND STANDARD DEVIATIONS FOR WALL-CHART AND ORTHO-RATER TESTS
(N = 117)

Target	Scoring Method	Mean		Standard Deviation	
		Wall Chart	Ortho-Rater	Wall Chart	Ortho-Rater
Letter (Test)	A	62.6	62.0	11.0	11.1
	B	64.8	64.2	11.0	11.5
	C	65.0	63.3	10.4	11.1
	D	68.9	66.3	10.8	11.8
Letter (Retest)	A	63.3	61.4	11.5	11.6
	B	65.6	63.7	12.2	12.0
	C	65.7	63.6	11.0	10.8
	D	69.6	67.6	11.5	11.2
Landolt (Test)	A	60.9	61.0	11.7	12.5
	B	62.3	63.0	12.0	12.8
	C	63.3	63.5	11.3	12.2
	D	66.8	67.5	12.0	12.9
Landolt (Retest)	A	62.0	62.7	11.3	13.2
	B	63.8	64.7	11.7	13.2
	C	64.3	65.1	11.3	13.7
	D	67.8	69.1	12.3	14.6

Ortho-Rater plates was 13.5 millilamberts. A MacBeth Illuminometer was employed in making light measurements. The required Ortho-Rater and wall-chart brightnesses were secured before each session, employing a voltmeter and a continuously variable resistance (variac).

The examiner observed the subject at all times to make sure that he did not squint or view the charts obliquely and that he was not fatigued. Responses were transmitted electrically to an adjacent room where they were checked by a technician and recorded on prepared answer forms.

The following presentation order of tests was maintained: wall-chart letter, wall-chart modified Landolt, Ortho-Rater letter, Ortho-Rater modified Landolt. These tests were a portion of a larger group of 17 mesopic and photopic targets given in the same session. Subjects had observed five mesopic wall charts and two mesopic Ortho-Rater plates before taking the four tests discussed here. The letter wall chart was the third test given on the photopic level, the modified Landolt wall chart was the fifth, the Ortho-Rater letter plate was the eighth, and the Ortho-Rater modified Landolt plate was the ninth of the 10 tests given at the photopic level.

The same procedure was followed in the retest session two weeks later.

RESULTS

An indication of the relative difficulty of wall-chart and Ortho-Rater presentation is shown in Table 1. The mean represents the average number of items achieved by the subjects before the criterion failure was met. The standard deviation is an indication of the magnitude of individual differences.

Results are presented for four scoring methods:

a. Number of rights before two consecutive miscallings were first made.

b. Number of items attempted before two consecutive miscallings were first made.

c. Number of rights before three consecutive miscallings were first made.

d. Number of items attempted before three consecutive miscallings were first made.

These methods were utilized to show the effect of scoring method on results and, thus,

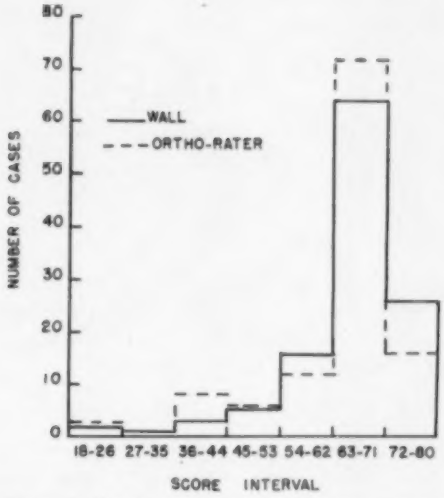

Fig. 2 (Gordon, et al.). Distribution of scores on the new Army Snellen tests. Items 30 to 39 of the tests are 20/20 acuity value. (N = 117.)

give the results wider generality. It should be noted that these scoring methods are non-independent measures.

It may seem that subjects were able to read further on the wall-chart letter tests than on the Ortho-Rater letter plates before meeting the criterion of failure. This difference in difficulty may perhaps be explained by the somewhat larger visual angle of the letter wall charts (see Method and Procedure). The scores on the Landolt tests, where more perfect reproduction of visual angle was achieved, are about equal for the two methods of presentation. The standard deviations are approximately the same, except that the Ortho-Rater Landolt retest shows greater variability than its wall chart.

Although there are several significant differences in means and standard deviations of the two methods of presentation, the differences are too small to be of practical importance. In Snellen acuity units, negligible changes in scores are implied. As shown in Figures 2 and 3, the test distributions are very similar. In general, the evidence does not indicate that Ortho-Rater and wall-chart presentations differ greatly in difficulty and variability.

The test-retest reliabilities of wall chart and Ortho-Rater scores are shown in Table 2. All Ortho-Rater reliabilities except Landolt ring, method A, are significantly higher than those of the wall charts.*

The higher reliabilities of the Ortho-Rater plates cannot be explained by the fact that the Ortho-Rater plates were administered after the wall charts. If increased reliability

* A significant difference implies that the difference found would be obtained less than once in 20 times by chance alone. Computation of significance involve tables of the t distribution (* p. 352).

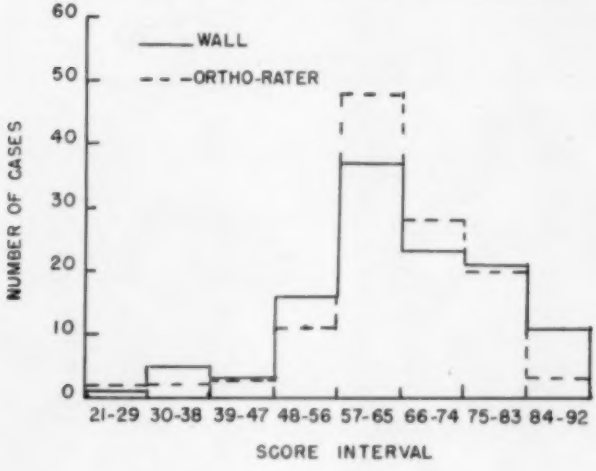

Fig. 3 (Gordon, et al.). Distribution of scores on the modified Landolt ring tests. Items 38 to 45 of the tests are 20/20 acuity value. (N = 117.)

TABLE 2

WALL-CHART AND ORTHO-RATER TEST RETEST RELIABILITIES
(N = 117)

Scoring Method	Letter Test		Landolt Test		A.F. Far Visual Acuity Test	
	Wall	Ortho-Rater	Wall	Ortho-Rater	Second	Tenth
A	0.81	0.90	0.73	0.81	0.90	0.83
B	0.78	0.89	0.69	0.79	0.88	0.81
C	0.88	0.92	0.75	0.85	0.81	0.86
D	0.80	0.87	0.65	0.79	0.80	0.81

is associated with later tests administered in the session, the Armed Forces far visual acuity test, administered twice, should have shown this effect. This test was administered as the second and 10th (last) test of the photopic series. The test administered in the last position shows a significant decrease in reliability for two scoring methods and a non-significant increase for two other scoring methods.

The correlations between scores on the wall charts and on the Ortho-Rater plates are presented in Table 3. These correlations are based on scoring method C, which was the most reliable method employed (see Table 2). They are about as high as the test-retest reliabilities. The mean of the correlations is equal to 0.83; the mean of the reliabilities of scoring method C is equal to 0.85.* The mean of the correlations, corrected for the attenuating effects of unreliability in each variable is 0.98.† These data offer little evidence to support the existence of a machine factor or "apparatus accommodation factor" specific to Ortho-Rater presentation.

DISCUSSION OF RESULTS

The finding that Ortho-Rater tests are more reliable than wall-chart tests presents a problem for interpretation. The superiority of instrument presentation may be due to lowered visual distraction with limitation of the surround, or to some other advantage of subject or stimulus characteristic leading to greater constancy of conditions. It is believed that the difference in reliability between Ortho-Rater and wall-chart presentation will be even greater in operational testing than that found here. It is well known that the conditions of wall-chart testing differ widely from place to place.

If visual angle, background luminance,

* The correlation of a test with another test will almost never be higher than the reliability of the test itself. The reliability of the measure limits the extent to which it can relate to another measure.

† The Spearman formula is employed in this computation (* p. 135). This formula gives an estimate of the correlation between variables with the attenuating effect of unreliability removed.

TABLE 3

CORRELATIONS* BETWEEN WALL-CHART AND ORTHO-RATER TESTS
(SCORING METHOD C)
(N = 117)

Variables	Test Session	Retest Session	O-R Test Session & Wall Retest Session	Wall Test Session & O-R Retest Session
Wall chart vs. Ortho-Rater (Letter)	0.85 (0.94)	0.87 (0.97)	0.86 (0.96)	0.89 (0.99)
Wall chart vs. Ortho-Rater (Landolt)	0.78 (0.98)	0.84 (1.00)	0.77 (0.96)	0.80 (1.00)

* Correlations corrected for attenuation are given in parentheses.

and contrast between object and background are equated as closely as possible between Ortho-Rater and wall-chart presentations, closely equivalent measures are obtained. The difficulties of the tests and the variabilities of scores are similar. When the correlations of the tests are taken into consideration, the methods appear to measure the same visual abilities.

Summary and Conclusions

This study presents a psychometric comparison of visual acuity scores obtained on Ortho-Rater plates with visual acuity scores on duplicate wall-chart tests. A comparison was made of the reliability of the two methods of presentation. An analysis was also made to determine whether or not both tests measured the same abilities. One hundred and 17 subjects were tested binocularly and retested two weeks later. Letter and modified Landolt ring targets were employed. Previous practice had been given on other mesopic and photopic wall charts and Ortho-Rater plates before the tests under consideration were given. The following results were obtained:

1. The two methods of presentation were of equal difficulty, except for slight discrepancies introduced by photographic reduction.

2. The reliabilities of the Ortho-Rater tests were significantly higher than those of the wall-chart tests.

3. The correlations between Ortho-Rater and wall-chart tests were about as high as the reliabilities of the test themselves. When corrected for attenuation, these correlations approach unity. No evidence is afforded, under these conditions, of a machine or "apparatus accommodation" factor affecting Ortho-Rater acuity scores.

Personnel Research Branch,
Department of the Army.

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Ophthalmic Miniature

A stronger remedy than these (for opacity of the cornea) is made by adding half a dram of the dung of the large green lizard called ligurus.

Ugo Benzi (1376-1439) *Consilium* 24

(Lockwood, Ugo Benzi, University of Chicago, 1951, page 145.)

THE PRINCIPLE OF THE BICENTRIC LENS IN ANISOMETROPIA

JOSEPH I. PASCAL, M.D.
New York

The correction of anisometropia entails two sources of possible discomfort for the patient: (1) Creation of unequally sized retinal images, and (2) creation of unequal prism effect when looking off center, as in reading.

It is reasonable to suppose that in order to produce discomfort the anisometropia must be of considerable extent. For certainly anisometropia of low degree, say, up to 1.0D. must be very common and biologically normal. The eyes as living structures are not cast from the same mould and slight anatomic and physiologic differences are to be expected. Marked anisometropia, however, say of 2.0D. and more, requires some special consideration.

A SPECIFIC INSTANCE

To take a specific instance. Suppose the right eye requires a +2.0D. and the left eye a +4.0D. lens. Monocularly, each lens is satisfactory but, when used binocularly, the lenses may produce some disturbances in binocular vision. Some patients learn to overcome these difficulties and adjust themselves after a brief period of mild discomfort. Others cannot adjust themselves, the lenses are uncomfortable, and a modification, usually a reduction of the stronger lens, has to be made.

The reason for the difficulties encountered centers mainly in the two factors already mentioned: (1) Differences in image size between the ocular images in the two eyes (aniseikonia), and (2) induced vertical prismatic imbalance obtained when looking off center, as in reading and other near vision tasks. It is generally agreed that the first—the creation of aniseikonia—is relatively unimportant in the majority of cases. The principal trouble is due to the second factor, the unequal prism effect.

PRISMATIC EFFECT MOST DISTURBING

Size difference really has to be measured. It cannot be assumed a priori because of the differences in lens power. If the ametropia is axial, and at least 50 percent of refractive errors are mainly axial, and if the correcting lenses are properly placed before the eyes, there may be no image size difference, or a difference of a minimal extent.

By properly placed, I mean placed so that P_2 (the second principal point) of the lens coincides with F_1 (the first principal focus) of the eye. In the ordinary meniscus, P_2 of the lens is generally some three mm. in front of the posterior surface of the lens. And if the latter is, say 12 mm. from the apex of the cornea, then P_2 of the lens will be about 15 mm. from the cornea, which is about the location of F_1 of the eye.

It is well known that image size differences even of considerable extent are often accepted without any complaints. So that this factor, as a cause of discomfort, is about the last one to be considered. However, unequal prism effect definitely has to be considered in every case.

EFFECT OF HEAD POSTURE

The difficulty encountered from unequal vertical prism effect, when looking below center, is variable. It depends very much upon the patient's habitual head posture when reading. Some persons tilt their head downward and use practically the central region of their lenses in near vision, as in distance vision. These persons have no difficulties from induced prism effects. Others, however, do not tilt their heads or do so only to a slight extent. They lower their eyes in reading and thus use in near vision a region below the optical center of the lens. These people naturally will have difficulties from the unequal prism effect.

The point below the optical center which is used for reading by these persons varies considerably but is generally taken to be somewhere between six and 10 mm. Let us take the mean value of eight mm.

In our example, eight mm. below center, the right lens has a prism effect of $\Delta = 0.8 \times 2 = 1.6^{\Delta}$ base-up. This is derived from the formula $\Delta = C \times D$ (read "delta equals C times D"). The left lens has $\Delta = 0.8 \times 4 = 3.2^{\Delta}$ base-up. The result is an excess of 1.6^{Δ} base-up over the left eye, creating a vertical imbalance of that amount.

Of course exactly the same thing happens with unequal-power concave lenses. Say, the right eye takes −2.0D. and the left eye takes −4.0D. At a point eight mm. below the optical center, the right lens has $\Delta = 0.8 \times 2 = 1.6^{\Delta}$ base-down, and the left lens has $\Delta = 0.8 \times 4 = 3.2^{\Delta}$ base-down, causing an imbalance of 1.6^{Δ} prism base-down over the left eye (left hypertropia of 1.6^{Δ}).

Slab-off prism principle

To equalize the prism effect at near, it is possible to grind the outer surface of the lenses so as to produce the same amount of prism power in this lens as in the other lens. What is really done is, in effect, to create two principal axes and two optical centers in the lens thus "slabbed" off. To understand the principle involved let us first study the mechanics in case of the minus lenses.

We saw that in the −4.0D. lens we get at a point eight mm. below the center 3.6^{Δ} prism (read 3.6 delta prism). The right lens has only 1.6^{Δ} prism at this point. To get only 1.6^{Δ} prism in the left −4.0D. lens (in order to match the prism in the right lens) the "decentration" must be only four mm.

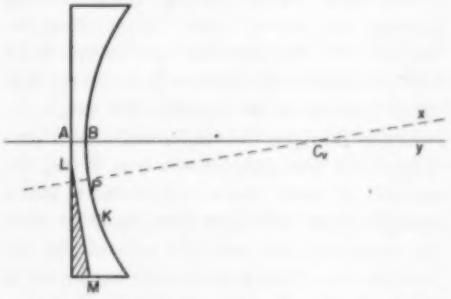

Fig. 1 (Pascal). Bicentric plano-concave lens.

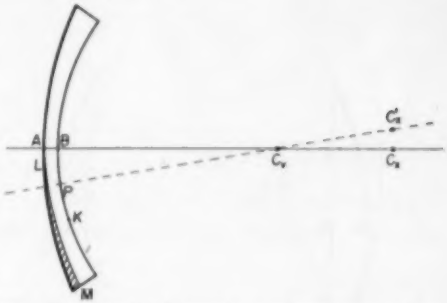

Fig. 2 (Pascal). Bicentric meniscus-concave lens.

From $\Delta = C \times D$, $1.6 = C \times 4$, $4C = 1.6$, $C = 0.4$, C stands for centimeters, therefore decentration $= 4.0$ mm. What we do now, in effect, is to introduce another optical center four mm. below the old optical center in the −4.0D. lens. We can see the application of this principle in the following diagrams.

Let us first consider a plano concave lens (fig. 1).

Bicentric plano-concave lens (fig. 1). C_v is the center of curvature of the inner concave surface. Y - C_v - B - A is the principal axis of the lens, the line perpendicular to both surfaces. The optical center of the lens lies on this line. At P, four mm. below B a plano surface L M is ground on the outside perpendicular to the line C_vP. This line perpendicular to the two surfaces of the lens is the principal axis of the lens *for the reading portion*. There are two principal axes in this lens, that is two lines perpendicular to the two surfaces of the lens, at different points on the lens. At point K, four mm. below P (and eight mm. below B), we get a prism of 1.6^{Δ} base-down, the same as in the right lens.

Let us examine now a concave meniscus, and see how the procedure works out here (fig. 2).

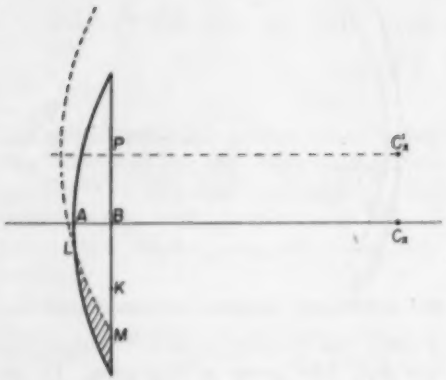

Fig. 3 (Pascal). "Slab-off" process in a plano-convex lens.

Bicentric meniscus-concave lens. (fig. 2). In Figure 2, C_x is the center of curvature of the convex surface, and C_xA is the radius of curvature of the convex surface. C_v is the center of curvature of the concave surface, and C_vB is the radius of curvature of the concave surface. $C_x - C_v - B - A$ is the principal axis of the lens on which is located the optical center. We want to grind the outer surface so as to have another principal axis four mm. below B at Point P. Imagine the grinding tool for the outer surface set so that the center of curvature C'_x is on a line connecting C_v and P. The outer surface below A follows the curve L M. The effect of this grinding is to place a new optical center on the line $C'_x - C_v - P$ which becomes the principal axis for the reading portion. The line is perpendicular to both surfaces, just as is line $C_x - C_v - B - A$, the principal axis for the distance portion. At point K, eight mm. below B, which is the point we assumed our patient uses for reading, he gets only 1.6$^\Delta$ prism base-down, since K is only 4 mm. from P and from the optical center on the axis through P.

Difference in procedure between plus and minus lenses

We equalized the prism effect in concave lenses at near by grinding down the stronger minus lens. In case of plus lenses, this is not a feasible procedure. What is done there is to grind down the weaker lens so as to produce a stronger prism effect to match the stronger prism effect of the stronger lens.

Let us take our example of O.D., +2.0D.: O.S., +4.0D. At a point eight mm. below center the right lens has 1.6$^\Delta$ prism base-up, the left lens has 3.2$^\Delta$ prism base-up. We now work on the right lens (the weaker lens) so as to produce 3.2$^\Delta$ prism at a point eight mm. below principal axis of the distance portion. To obtain 3.2$^\Delta$ with a +2.0D. lens requires a "decentration" of 16 mm. (from $\Delta = C \times D, 3.2 = C \times 2, 2C = 3.2, C = 1.6$ cm. = 16 mm.). We must therefore place the new optical center for the reading portion 16 mm. from point K (fig. 3) and therefore eight mm. *above* B.

Let us consider first a plano-convex lens, Figure 3.

Slab-off process in a plano-convex lens (fig. 3). C_x is the center of curvature of the outer convex surface. $C_x - b - A$ is the principal axis, perpendicular to both surfaces. We now create another principal axis at P eight mm. *above* B by placing the center of curvature of the convex surface C'_x on the perpendicular to the plano surface at P. The spherical curve created with C'_x as the new center of curvature cuts the lower part of the lens at L M. The direction of this curve above L is indicated by the dotted line. Thus point K is 16 mm. from the optical center of the curve corresponding to the reading portion, the optical center lying along the line $C'_x - P$. We therefore get at this point 3.2$^\Delta$ prism base-up which will match the 3.2$^\Delta$ prism base-up of the stronger left lens.

Let us now see the mechanics of this procedure in a meniscus convex lens. Here, too, we have to place a new optical center and a new principal axis eight mm. *above* the optical center and the principal axis of the distance portion. This requires another principal axis at P (fig. 4) eight mm. above B.

Slab-off process in a meniscus convex (fig. 4). C_x is the center of curvature of the outer convex surface, C_v is the center of curvature of the inner concave surface (longer radius for surface of less power). Line $C_v - C_x - B - A$ is the principal axis of the lens, perpendicular to both surfaces. At a point P, eight mm. above B, draw line $C_v - P$, that is a line through the center of curvature of the concave surface and the point P where the new principal axis will cut the lens. Place C'_x the new center of curvature on this line so as to cut the outer surface at L M. $C_v - C'_x - P$ is the new principal axis for the reading portion, and a new optical center for the reading portion is located on this line. At point K, eight mm. below B, we have a decentration of 16 mm. and a prism effect of 3.2^{Δ} to match the prism effect of the right lens.

It will be seen that each lens so marked has two principal axes and two optical centers, one for the distance portion, one for the lower reading portion, though the lens has only *one power*, that is, the same power at the reading sector as at the distance sector. The lens is therefore appropriately called a bicentric lens.

The procedure just discussed is used more generally to equalize the prism effects in bifocal lenses. The principle is essentially the same. Of course this discussion is designed to explain how the effects are produced. The actual, mechanical process of grinding, as carried out by the grinder mechanic, does not show the principle involved so readily.

In some special lenses, for example plano-meniscus—that is plano in power, meniscus in shape—the slab-off prism method is used to incorporate prism power in the lower part of the lens, generally in connection with a bifocal segment. Such a lens may not have two principal axes or be bicentric. But here, too, the process can be explained by a shift of the center of curvature of the convex surface.

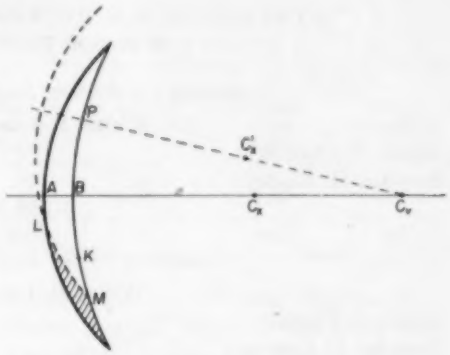

Fig. 4 (Pascal). "Slab-off" process in a meniscus convex.

Summary

An ingenious method of grinding a lens so as to have two principal axes and two optical centers at different sectors of the lens has now been devised. Its purpose is to equalize the prismatic effects in anisometropic lenses at the reading level in single-power lenses and in bifocal lenses. The lenses are known as bicentric lenses, and the process referred to as the "slab-off-prism" procedure.

An explanation of the principle involved by visualizing a shifting of the center of curvature of the grinding tool for the outer surface is here presented.

The same principle is shown to be effective in both convex and concave lenses with this important difference. In convex lenses the weaker lens is "slabbed" off so as to produce a stronger prism effect in order to equalize the prism effect in the stronger lens. In concave lenses the stronger lens is "slabbed" off so as to produce a weaker prism effect. The illustrative diagrams help visualization of the procedure and the principle involved.

41 West 96th Street (25).

STANDARDS FOR REFERRAL OF SCHOOL CHILDREN FOR AN EYE EXAMINATION

Committee for the New England Ophthalmological Society

Walter B. Lancaster,* *chairman*

Edwin B. Dunphy — James J. Regan
Brendan D. Leahey — Albert E. Sloane
Elton R. Yasuna

Committee for the Massachusetts Medical Society

Walter B. Lancaster,* *chairman*

Edwin B. Dunphy — James J. Regan
Brendan D. Leahey — Albert E. Sloane
Elton R. Yasuna

Committee for the Harvard School of Public Health and The Children's Medical Center

Harold C. Stuart — J. Roswell Gallagher — William Schmidt

In recent years the subject of vision testing in the schools has been given considerable attention. The early detection of those pupils whose defective vision could adversely affect their physical or scholastic efficiency has been encouraged by both physicians and educators, and research has been devoted to the development of more sensitive and more efficient screening devices. .

However, the question of what constitute the ideal standards for referral still remains unanswered. Until this matter can be clarified, it will remain difficult to estimate the relative efficiency of the various screening methods now in use. It is obviously the intent to so construct screening devices that *all* pupils requiring a specialist's examination will be detected and *very few* will be needlessly referred.

The screening device and the school's eye health program both lose prestige when pupils needing attention are not screened out or when many of those sent to a specialist return with a report that the referral was unnecessary. Yet such conditions will continue, even though screening devices and the skill of testing personnel are improved, unless differences in standards for referral can be reconciled.

It would seem clear that it is the responsibility of the ophthalmologists to agree upon, and to set, standards for referral which they, as a group, feel to be optimal.

This report reviews the preliminary findings of a study designed to attack the general problem of suitable standards for referral. By use of a questionnaire information regarding standards which physicians use in their own offices and standards which they advocate that others use for referral has been obtained and analyzed. Questions regarding their choice of screening test, who should administer the test, and who should refer the pupil were also included.

Method

The content of the questionnaire, which had reference only to patients between the ages of six and 18 years, is disclosed under the section, *Results,* which follows.

A copy was sent to all physicians who were either members of the New England Ophthalmological Society or who were Fellows of the Massachusetts Medical Society's

* Deceased.

TABLE 1

RESPONSE RATES BY MEDICAL SPECIALTY AND LENGTH OF PRACTICE

Length of Practice	Full-Time Questionnaires Mailed	Ophthalmologists Responding (percent)	Others		Total	
			Questionnaires Mailed	Percent Responding	Questionnaires Mailed	Percent Responding
Less than 20 yr.	79	65	71	61	150	63
20 yr. or more	61	49	89	27	150	36
Total	140	58	160	42	300	50

Section of Ophthalmology and Otolaryngology and who practice ophthalmology. It was requested that the questionnaires *not* be signed; in order to determine the names of those who had not returned a questionnaire, each physician who filled one out was requested to return a separate postcard. This latter device allowed us to obtain information on the type and length of practice of those who returned questionnaires (table 1).

Subsequently a smaller survey was conducted among a group of 14 ophthalmologists selected because of their special interest in the problems of school vision testing. All of this group returned questionnaires: their replies are discussed under Part II.

PART I: RESULTS

Questionnaires were mailed to 300 physicians. Fifty percent of these (149) completed and returned questionnaires. (This group of 149 does not include the 14 ophthalmologists who belonged to the small special survey already referred to.) On the basis of information in the *American Medical Directory*, all 149 physicians were classified by medical specialty and by length of practice (table 1). It will be seen that the highest response rate (65 percent) occurred in the full-time ophthalmologists who have practiced less than 20 years. The lowest response rate (27 percent) was from those who do not devote full time to ophthalmology and who have practiced 20 or more years. A part of the low response rate among other than full-time ophthalmologists can be explained by the fact that five of these physicians returned cards saying they limited their practice to a field other than ophthalmology and six other questionnaires were unanswered because of retirement or death.

FIRST PART OF QUESTIONNAIRE

The percent distribution of replies to each of the questions relating to the physician's procedure *in his own office practice* was as follows:

1. What visual acuity (in the worse eye) do you ordinarily consider deserving of your consideration for treatment? Encircle the *highest* visual acuity to which you *usually* give attention and treatment. See Table 2.

In their own office practice about 35 percent of this group usually give attention and treatment to children of from six to nine years of age who have 20/30 vision in one eye, and to young people of 10 to 18 years of age who have 20/25 vision in the poorer eye.

2. How much hypermetropia (elicited without cycloplegia) in the worse eye and

TABLE 2

VISUAL ACUITY NEEDING TREATMENT

	20/25	20/30	20/40	20/50	20/100
	(percent)	(percent)	(percent)	(percent)	(percent)
a. At ages 6–9 yr.	9.0	36.1	47.2	5.6	2.1
b. At ages 10–18 yr.	35.7	47.5	14.0	1.4	1.4

TABLE 3

HYPERMETROPIA NEEDING TREATMENT

DIOPTER SPHERES	+1.00	+1.50	+2.00	+2.50	+3.00	+3.50	+4.00
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. At Ages 6–9 yr.							
Total group	7.6	29.9	28.5	13.9	13.2	2.0	4.9
Full-time ophthalmologist	7.6	25.5	26.4	16.0	16.0	2.8	5.7
Others	7.9	42.1	34.2	7.9	5.3	—	2.6
b. At Ages 10–18 yr.							
Total group	30.3	40.0	15.9	8.3	4.1	0.7	0.7
Full-time ophthalmologist	26.7	37.1	19.1	10.5	4.8	0.9	0.9
Others	40.0	47.5	7.5	2.5	2.5	—	—

not associated with a significant muscle imbalance or symptoms of asthenopia is ordinarily deserving of your consideration for treatment? Encircle the *least* amount to which you usually give attention under those conditions. See Table 3.

A comparison of the replies from full-time ophthalmologists with those from other physicians has been included in this section. The full-time ophthalmologist apparently tends to require a greater degree of hypermetropia before he recommends treatment, but the difference between the two groups is not great: 36 percent of the full-time group require two or more diopters (at ages 10 to 18 years) whereas only 29 percent of the others require that much.

3. How many prism diopters of esophoria are ordinarily deserving of your consideration for treatment? Encircle the *least* amount. See Table 4.

A considerable difference in the office practice of these physicians shows up here. In regard to esophoria (distant) 24 percent of these physicians consider treatment when four diopters of esophoria are present, and about an equal number do not seriously regard this condition unless 10 or more diopters are present.

4. How many prism diopters of exophoria are ordinarily deserving of your consideration for treatment? Encircle the *least* amount. See Table 5.

Similarly about 20 percent regard four diopters of exophoria (distant) as deserving consideration for treatment and about the same number do not seriously regard less than 12 diopters.

5. How many prism diopters of hyperphoria are ordinarily deserving of your consideration for treatment? Encircle the *least* amount. See Table 6.

As one would expect, there is a narrower range of limits here, but there were a few instances of wide variance from the majority opinion.

6. If heterotropia is not present, do you routinely use cycloplegia in refracting *children?* Encircle one:

YES	NO
77.8 percent	32.2 percent

7. If your answer to question 6 is "yes," please encircle the age limit at which you begin to use a cycloplegic routinely:

TABLE 4

ESOPHORIA NEEDING TREATMENT

PRISM DIOPTERS	2	4	6	8	10	12	14	16
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. Distance	2.8	21.7	30.7	20.3	17.5	3.5	1.4	2.1
b. Near	5.1	24.1	21.2	24.8	13.9	6.6	3.6	0.7

TABLE 5

EXOPHORIA NEEDING TREATMENT

PRISM DIOPTERS	2	4	6	8	10	12	14	16	18	20
	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)	(per-cent)
a. Distance	3.5	16.0	25.7	17.4	19.4	6.2	6.2	2.1	2.8	0.7
b. Near	2.8	17.5	9.8	17.5	20.2	14.0	7.7	8.4	1.4	0.7

TABLE 6

HYPERPHORIA NEEDING TREATMENT

PRISM DIOPTERS	1	1.5	2	2.5	3	4	6	8
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. Distance	19.7	27.9	40.2	2.7	7.5	2.0	—	—
b. Near	21.2	30.8	36.3	2.1	7.5	2.1	—	—

(Omitted from analysis because of ambiguous wording.)

NOTE: The replies to this question have been discarded. The question was poorly phrased and its intent misinterpreted by many who attempted to answer it.

8. Do you *routinely* test for heterophoria? Encircle one:

	YES (percent)	NO (percent)
Total group	89.8	10.2
Full-time ophthalmologists	96.3	3.7
Others	71.8	28.2

9. If your answer to question 8 is "yes," encircle the test you employ. See Table 7.

10. Do you *routinely* test for any of the following? Encircle the tests you routinely employ. See Table 8.

11. Do you limit your practice to ophthalmology? Encircle one:

YES	NO
73.2 percent	26.8 percent

This question permitted us to make a separate analysis of the replies from full-time ophthalmologists.

TABLE 7

HETEROPHORIA TESTS USED

	Horizontal Alone	Vertical Alone	Horizontal and Vertical
	(percent)	(percent)	(percent)
a. Distance			
Total group	5.3	3.1	91.6
b. Near			
Total group	16.1	5.1	78.8
Full-time ophthalmologits	11.7	3.2	85.1
Others	33.3	12.5	54.2

TABLE 8

ROUTINE TESTS EMPLOYED

	Total Group	Full-Time Ophthalmologists	Others
	(percent)	(percent)	(percent)
P. P. convergence	94.8	100.0	80.0
Amplitude of fusion	25.9		
Stereopsis	41.4	51.2	13.3
Color vision	30.2		

TABLE 9

VISUAL ACUITY FOR REFERRAL

VISION	20/25	20/30	20/40	20/50	120/100
	(percent)	(percent)	(percent)	(percent)	(percent)
a. At ages 6–9 yr.	4.0	39.6	50.4	4.7	1.3
b. At ages 10–18 yr.	33.6	48.3	16.1	1.3	0.7

SECOND PART OF QUESTIONNAIRE

The second part of the questionnaire related to these same physicians' opinions in regard to *tests given in schools.*

1. Do you consider symptoms suggestive of eye discomfort reason enough to refer a school child to you for an eye examination?

YES	No
94.6 percent	5.4 percent

2. Do you consider the development of a hitherto unnoticed eye defect reason enough for a teacher to refer a child to you for an eye examination? Encircle one:

YES	No
99.3 percent	0.7 percent

3. Should a child who has difficulty in learning to read but who has no subjective ocular symptoms and normal visual acuity on a school test be referred for an eye examination? Encircle one:

YES	No
87.2 percent	12.8 percent

4. At what visual acuity (in the worse eye) would you like a school child referred to you for further investigation? Encircle the highest visual acuity you would like referred. See Table 9.

As one would expect the referral level is somewhat lower than the level set for consideration for treatment in office practice (see also Table 14). Forty-eight percent of these physicians would like young people (age 10 to 18 years) referred to them when the poorer eye sees no better than 20/30.

5. Do you believe a test for hypermetropia should be made in the schools? Encircle one:

YES	No
62.9 percent	37.1 percent

6. Whether your answer to question 5 is "yes" or "no," how much hypermetropia *in the worse eye* elicited without cycloplegia in a school test should be reason for referral of the child to you for further investigation? Encircle the least amount you would like referred. See Table 10.

Here again there is a tendency to suggest a lower level for referral than the one used in office practice (see also Table 14). However, about 30 percent of physicians stated that they do not wish pupils (age 10 to 18 years) showing less than two diopters of hypermetropia referred to them; an equal number would set one diopter to be the least amount for referral.

7. Do you believe tests for heterophoria should be made in the school? Encircle one:

YES	No
46.6 percent	53.4 percent

8. Whether your answer to question 7 is "yes" or "no," how many prism diopters of esophoria elicited in a school test would you consider sufficient reason for referral to you for further investigation? Encircle the least amount you would like referred. See Table 11.

TABLE 10

HYPERMETROPIA FOR REFERRAL

DIOPTER SPHERES	+1.00	+1.50	+2.00	+2.50	+3.00	+3.50	+4.00
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. At ages 6–9 yr.	10.4	28.5	34.0	14.6	9.0	2.8	0.7
b. At ages 10–18 yr.	31.2	38.9	20.8	5.6	2.8	0.7	—

TABLE 11

ESOPHORIA FOR REFERRAL

PRISM DIOPTERS	2	4	6	8	10	12	14	16
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. Distance	5.8	20.5	27.0	19.0	16.8	4.4	5.8	0.7
b. Near	12.0	13.5	25.6	19.6	16.5	9.0	2.3	1.5

About an equal number (26 percent) of physicians would like to have pupils having four diopters of esophoria (distant) referred as would set the limit at 10 or more diopters.

9. Whether your answer to question 7 is "yes" or "no," how many prism diopters of exophoria elicited in a school test would you consider sufficient reason for referral of the child to you for further investigation? Encircle the least amount you would like referred. See Table 12.

We have included here (under "b") a comparison of the replies from full-time ophthalmologists with those of the other physicians. As in their office practice, there is a wide difference of opinion among members of both groups. Seventeen percent of the full-time ophthalmologists would like pupils showing as little as four diopters of exophoria (near) referred to them and about 24 percent of the same group would not set the referral level at 14 or more diopters.

10. Whether your answer to question 7 is "yes" or "no," how many prism diopters of hyperphoria elicited in a school test would you consider sufficient reason for referral of the child to you for further investigation? Encircle the least amount you would like referred. See Table 13.

11. In a school screening test should the error in the number of referrals to you be in the direction of referring too many or too few? Encircle one:

TOO MANY	TOO FEW
85.8 percent	14.2 percent

12. Do you believe that to do the Snellen Test alone is sufficient in a School Vision Screening Test? Encircle one:

YES	NO
34.5 percent	65.5 percent

13. How frequently should routine School Visual Testing be done? Encircle one:

EVERY SIX MONTHS
12.2 percent

EVERY YEAR
70.1 percent

EVERY TWO YEARS
17.7 percent

14. Whom do you believe should administer the routine School Visual Screening Test? Encircle *one* or *more:*

	PERCENT
Teacher who has been given special instruction in eye testing	57.0
Teacher	17.4
School nurse	59.7
Ophthalmologist	38.9
School physician	29.5
Optometrist	18.8

TABLE 12

EXOPHORIA FOR REFERRAL

PRISM DIOPTERS	2	4	6	8	10	12	14	16	18	20
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. Distance										
Total group	5.8	19.6	21.8	10.9	20.3	10.9	7.2	1.4	0.7	1.4
b. Near										
Total group	5.9	14.0	11.7	15.4	20.6	13.2	10.3	5.9	1.5	1.5
Full-time ophthalmologists	5.0	11.9	9.9	13.8	21.8	13.8	12.9	6.9	2.0	2.0
Others	8.6	20.0	17.1	20.0	71.1	11.4	2.9	2.9	—	—

TABLE 13

HYPERPHORIA FOR REFERRAL

PRISM DIOPTERS	1	1.5	2	2.5	3	4	6	8
	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)	(percent)
a. Distance	19.4	26.6	38.1	5.8	7.2	2.2	—	0.7
b. Near	19.1	29.4	39.8	1.5	5.9	2.9	0.7	0.7

Lay technician who has been given special instruction in eye testing	57.7

15. Whom do you believe should recommend referral? Encircle *one* or *more:*

	PERCENT
Teacher who has been given special instruction in eye testing	53.7
Teacher	31.6
School nurse	69.1
Ophthalmologist	45.6
School physician	54.5
Optometrist	24.8
Lay technician who has been given special instruction in eye testing	53.7

PART II: REPLIES FROM QUESTIONNAIRES SENT TO A SMALL (14) SELECTED GROUP OF OPHTHALMOLOGISTS

All 14 questionnaires were returned from the group of physicians who are known to be closely associated with the problems of school vision testing. In general, the replies showed no significant difference from those of the larger group except in the tests for hypermetropia and exophoria. In each of these the members of this group tended to have more selective referral levels.

The majority of these respondents approved referral of a school child on the basis of symptoms suggestive of eye discomfort, the development of a hitherto unnoticed eye defect, or because of difficulty in learning to read. All agreed that school screening procedures should err in the direction of referring too many children. Most of this group favored annual screening examinations and regarded the Snellen test alone to be insufficient for school testing. The majority recommended the inclusion of

TABLE 14

PERCENT DISTRIBUTION OF OPINIONS WITH REFERENCE TO REFERRAL LEVELS FOR SCHOOL VISION TESTS

Test	Referral Level	Percent of Replies Favoring:		
		A Lower Level	This Level	A Higher Level
Visual Acuity				
At ages 6–9 yr.	20/40	44	50	6
At ages 10–18 yr.	20/30	34	48	18
Hypermetropia				
At ages 6–9 yr.	+2.0D. sph.	39	34	27
At ages 10–18 yr.	+1.5D. sph.	31	39	30
Esophoria				
Distance	6 P.D.	26	27	47
Near	6 P.D.	25	26	49
Exophoria				
Distance	8 P.D.	47	11	42
Near	10 P.D.	47	21	32
Hyperphoria				
Distance	2 P.D.	46	38	16
Near	2 P.D.	48	40	12

a test for hypermetropia but no more than half favored testing for heterophoria.

Discussion

On the basis of question 11 of Part I: "Do you limit your practice to ophthalmology?", the questionnaires of the full-time ophthalmologists were analyzed separately from the others. In general, there was a high degree of agreement between the answers given by these two groups of respondents. Differences which were statistically significant at the five-percent level were noted in only five of the 25 questions analyzed.

Most of these significant differences concerned the tests used in office practice. The full-time ophthalmologists reported significantly more routine use of tests for heterophoria, P.P. convergence, and stereopsis than did the other respondents. In the case of other testing procedures, the differences found, although not statistically significant, were also in the direction of more routine use by full-time ophthalmologists. A significant difference was also found in the amount of hypermetropia which is ordinarily deserving of consideration for treatment. Full-time ophthalmologists tended to require more hypermetropia before considering treatment.

Similarly, when asked about exophoria (near) as measured in a school test, the full-time ophthalmologists showed a significant tendency to recommend higher levels of referral. In most of the remaining questions about levels at which patients should be referred or considered for treatment, the full-time ophthalmologists continued to show a slight, but nonsignificant, tendency to favor higher, more selective, cut-off values.

The majority of the respondents approved referral of a school child for eye examination because of the following conditions:

1. Symptoms suggestive of eye discomfort (95 percent).
2. Development of a hitherto unnoticed eye defect (99 percent).
3. Difficulty in learning to read without subjective ocular symptoms and with normal visual acuity on a school test (87 percent).

There was also general agreement (86 percent) that school screening procedures should err in the direction of referring too many rather than too few children.

Beyond these general principles of referral, there was less general agreement concerning the details of the testing procedure that should be used in the schools. A clear majority (70 percent) favored annual examinations and most of the respondents (66 percent) felt that the Snellen test alone was insufficient for school testing. A school test for hypermetropia was recommended by a majority (63 percent) but slightly less than half (47 percent) of the respondents were in favor of a school test for heterophoria.

It should be noted that if the 149 individuals who answered the questionnaire are regarded as a random sample of a much larger group of ophthalmologists, the random sampling variation is such that a finding of 58 percent to 60 percent is necessary in order to establish a statistically significant majority opinion. Therefore, the results concerning the use of tests for heterophoria should be regarded as somewhat ambiguous. There is not clear evidence indicating a majority either for or against this particular test.

In the questions dealing with the referral levels for different kinds of school vision tests, there was much variation in opinion. Table 14 summarizes these findings. Inspection of Table 14 will show that the referral levels selected are those for which there was neither a majority favoring a higher level nor a majority favoring a lower level. In several instances, however, the proportion favoring a shift in one direction or another is so large that there is statistical uncertainty as to the nature of the majority opinion.

Finally, opinions as to who should give school vision tests and who should recommend referral yielded somewhat ambiguous results. Majorities, of borderline significance,

TABLE 15

COMPARISON OF "CONSIDERATION FOR TREATMENT" LEVELS USED IN OFFICE PRACTICE WITH REFERRAL LEVELS RECOMMENDED FOR SCHOOL TESTING

Test	Percent of Replies Indicating:		
	Office Level Lower than Referral Level (under referral)	Office Level Same as Referral Level	Office Level Higher than Referral Level (over referral)
Visual Acuity			
At ages 6–9 yr.	20	65	15
At ages 10–18 yr.	13	77	10
Hypermetropia			
At ages 6–9 yr.	18	49	33
At ages 10–18 yr.	21	52	27
Esophoria			
Distance	28	55	17
Near	23	55	22
Exophoria			
Distance	20	57	23
Near	20	56	24
Hyperphoria			
Distance	14	74	12
Near	18	72	10

endorsed the teacher with special training (57 percent), the school nurse (60 percent), and the lay technician with special training (58 percent) as individuals who should administer routine school vision tests.

A clear majority (69 percent) said the school nurse should recommend referrals, while nonsignificant majorities said that this function could also be performed by the teacher with special training (54 percent), the school physician (55 percent) and the lay technician (54 percent). A near majority (46 percent) said that an ophthalmologist should recommend referral after school vision tests.

In spite of the strong preference expressed in favor of over-referral by school testing programs, there was very close agreement between the "consideration for treatment" levels reported for office practice and the referral levels recommended for school tests. Table 15 indicates the relationship between these two levels for each of the tests.

In several instances Table 15 appears to show a slight (but not significant) tendency to favor under-referral rather than over-referral. In general, however, the suggestion is that the respondents did not distinguish systematically or consistently between levels of consideration for treatment in the office and school referral levels.

SUMMARY

A preliminary survey of physicians by the questionnaire method has indicated that a considerable difference of opinion exists in regard both to their office practice and to their desires in regard to the standards which they would set for the referral of school children to them.

This preliminary survey suggests that a further study of the problem of referral standards is desirable. In order to improve the efficiency of present vision screening methods and to improve their standing with the public as well as with practicing ophthalmologists a clarification and better understanding of the present situation is necessary.

VISUAL FIELD EXAMINATION BY A NEW TACHYSTOSCOPIC MULTIPLE-PATTERN METHOD

A PRELIMINARY REPORT

DAVID O. HARRINGTON, M.D., AND MILTON FLOCKS, M.D.
San Francisco, California

The measurement of extrafoveal visual acuity is of utmost importance in the evaluation of disease of the eye and the brain. Perimetry is now an established routine in the office of the ophthalmologist, neurologist, and neurosurgeon. Even the internist utilizes the confrontation method of field study as part of his physical examination.

Because the standard techniques of perimetry are tedious and time consuming and require the services of a skilled technician or medical perimetrist it is a relatively neglected method of examination. It requires rather elaborate equipment and, usually, a special room and is therefore generally unsuited to bedside practice where field examination is almost totally neglected.

In almost all of the screening methods of examination of visual function, such as those used in the public school system, in testing for driver's license and in industry, the examination of the field of vision is ignored because its complexity makes it impractical.

The insidious onset and high incidence of glaucoma has made this disease a public health problem of considerable importance. A routine screening method of visual field examination of all persons over 40 years of age might reduce the incidence of visual field loss by detection of the disease in its early stages.

The integrity of the visual field of the drivers of today's high-speed motor cars is a matter which greatly concerns the safety of everyone on the highway, yet adequate central visual acuity only is demanded to obtain a license. Individuals with "tunnel" vision or homonymous hemianopia can and do pass these tests.

Industrial screening methods should certainly strive to detect visual field loss in those working with or near moving machinery, overhead cranes, and the like.

DESCRIPTION OF TECHNIQUE

We have undertaken the development of a quick and simple method of qualitative visual field examination which can be used as part of a visual screening test or preliminary to a more detailed perimetric study.

The method, which consists of the rapid presentation of abstract patterns in the field after the manner of the tachystoscopic training course used by the Air Force to teach airplane recognition, can be performed and intrepreted by an unskilled technician. Each pattern is designed to detect a certain type of visual field defect and there are nine patterns so that the majority of defects are effectively encompassed.

The method is not intended to supplant present methods of quantitative tangent-screen and arc perimetry. It is a rapid qualitative examination which might be compared to the Ishihara test for color vision.

The patterns are printed in white fluorescent sulfide ink on 14 by 18-inch white cards and the fixation point is a five-mm. black dot. In ordinary illumination only the fixation point is visible, the rest of the card appearing plain white.

When the card is exposed to a source of "black light" from a Harrington Lumilamp[1] or other ultra-violet radiation, the pattern appears as a series of luminous lines or dots against a purple background. Fixation is maintained on the black dot while the card is exposed to the "black light" for 0.2 or 0.3 seconds and the patient then tells what he has seen. Patterns are simple and easily described.

Fig. 1 (Harrington and Flocks). Tachystoscope in operation, showing position of pattern cards.

Each eye is tested separately with each card but, as will be shown later, it may be advantageous to perform the test with both eyes open to elicit an extinction phenomenon which may be present both monocularly and binocularly (fig. 1).

The cards are best exposed in a box with a chin rest mounted on the opened lid so that the eye is 330 mm. from the fixation spot. The black light is mounted on the box lid under the patient's chin and equipped with a shutter so that the exposure time may be easily controlled. For the practical application of the examination we have not found that a timing device was necessary. As a means of making the test a more quantitative one, it will be used in future experiments.

The examination may be carried out in ordinary room light but is best performed when the light is slightly dimmed. There must be sufficient light to enable the patient to fix accurately before exposure of the pattern to the black light (fig. 2).

Fig. 2 (Harrington and Flocks). Tachystoscope in operation, showing source of black-light illumination and shutter.

Fig. 3 (Harrington and Flocks). *Pattern 1.* A test pattern to check fixation and distance from eye to card.

Patterns used

The following patterns have been used to date although it is probable that there will be others which will prove of value as experience is gained in the test.

Pattern 1 (fig. 3). The small crosses are 15.5 degrees distance from fixation, which is the location of the average blindspot. When one eye is closed, the temporal cross should disappear completely. If it does not it is either because the distance from the patient's eye to fixation is incorrect or that the patient is not fixing accurately on the central dot. This is a test card for fixation and distance and to acquaint the patient with the nature of the examination.

Pattern 2 (fig. 4). This pattern has four vertical lines, two at five degrees and two at 20 degrees from fixation. It is especially use-

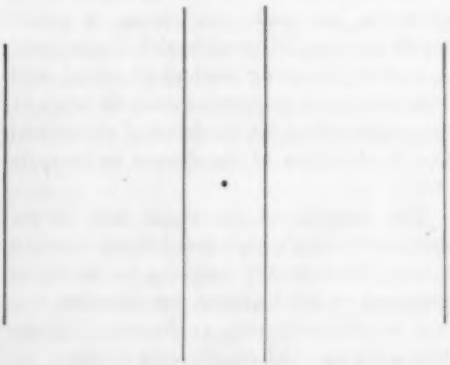

Fig. 4 (Harrington and Flocks). *Pattern 2.* Especially useful in the detection of hemianopic defects.

Fig. 5 (Harrington and Flocks). *Pattern 3.* For use with left eye and useful in detection of sector defect and enlargement of the blindspot.

ful in detecting hemianopic field defects, either homonymous or bitemporal.

Pattern 3 (fig. 5). This pattern is for the left eye only. The cross is somewhat larger than the normal blindspot so that a part of it should be normally seen. Failure to see the upper nasal oblique line will indicate a defect in this area.

Pattern 4 (fig. 6). The same as Pattern 3 but for the right eye.

Pattern 5 (figs. 7 and 8). An abstract design calculated to detect any visual field loss in the area of the lines. Especially useful in chiasmal lesions and occasionally in glaucoma.

Pattern 6 (figs. 7 and 8). The same as Pattern 5 inverted.

Pattern 7 (fig. 9) is made up of concentric

Fig. 6 (Harrington and Flocks). *Pattern 4.* Same as Pattern 3 but for use with right eye.

Figs. 7 and 8 (Harrington and Flocks). *Pattern 5.* Abstract design especially useful in chiasmal lesions and in glaucoma. *Pattern 6.* The same as Pattern 5 inverted.

circles at one, five, and 20 degrees from fixation. Paracentral and central scotomas are detected with this pattern. Breaks in the circles may be described in certain glaucomatous field changes. Hemianopic field defects may be found but one must be careful that the "completion phenomen" described by Bender[2] does not confuse the result.

Fig. 9 (Harrington and Flocks). *Pattern 7.* Concentric circles for detection of scotomas and certain glaucoma defects.

Fig. 10 (Harrington and Flocks). *Pattern 8*. Especially useful in the detection of the nerve-fiber bundle defect of glaucoma.

Pattern 8 (fig. 10). Four vertical dots are arranged two above and two below fixation at five degrees and 10 degrees. Particularly designed to detect the nerve-fiber bundle defect of glaucoma.

Pattern 9 (fig. 11). Four dots are arranged in the four quadrants of the field at a distance of 15 degrees from fixation. This pattern is of particular value for binocular testing of hemianopic field defects where "extinction" is a factor. Also of value in sector defects from whatever cause.

The last card is dull black with a central red dot printed in fluorescent-sulfide ink. It is used with the Harrington lumitest[1] objects as a portable tangent screen for use where complete perimetric equipment may not be readily available. Used with a one-mm. luminescent test object it is quite accurate and is particularly useful for bedside perimetry. When certain of the patterns have been incorrectly described, indicating a visual field defect, the area in question may be quickly explored on the black card, thus, in effect, completing the perimetric examination in a quantitative manner.

Any large or dense visual field defect is easily and quickly detected by exposure of the pattern cards. Retinal detachments, hemianopias, scotomas, sector defects, and moderately large glaucomatous defects make it difficult or impossible to read the patterns correctly and suggest the nature of the defect in each case.

Comment

Certain questions have arisen in the development of this method of perimetry which will require more experience and more clinical data to answer:

1. Should the lines be made thicker or thinner or should they be dotted lines?
2. Are the patterns too complicated?
3. Are more or fewer patterns desirable?

Bender[2] has demonstrated that a homonymous hemianopia, so slight as to be undetectable with ordinary single stimulation,

Fig. 11 (Harrington and Flocks). *Pattern 9*. Of value in the detection of sector or hemianopic defects and in testing hemianopic defects where "extinction" is a factor.

Fig. 12 (Harrington and Flocks). Tachystoscope folded for carrying.

may be easily found when multiple stimulation is used; the stimulus in the "seeing" field serving to "extinguish" the stimulus in the anopic field.

In the pattern method, each stimulus acts to lessen the effective size of every other stimulus, particularly in the presence of retinal or cerebral disease. Thus a hemianopia which may not be found with very small test objects by ordinary methods of perimetry can sometimes be demonstrated by the tachystoscopic pattern method because of "extinction."

In everyday life, the individual is constantly receiving multiple visual stimuli from every direction. When used monocularly and binocularly with multiple stimuli, the pattern test may be a better gauge of extrafoveal visual function than ordinary perimetric methods.

In this regard it is our clinical impression that in elderly and arteriosclerotic individuals "extinction" is a common phenomenon when multiple stimuli are used. That is why they do poorly in the pattern test when their visual fields by conventional perimetry are within normal limits.

Summary

1. A new method of visual field examination is presented using a tachystoscopic exposure of various patterns each designed to detect a specific visual field defect.

2. The patterns are printed in fluorescent sulfide ink and become visible only when exposed to the ultraviolet radiation of a "black light" tube.

3. The method may be used as a screening test which can be performed on large numbers of individuals by relatively untrained examiners. It should be of value in testing personnel in industry and applicants for driver's licenses, and as part of the visual screening test in adult schools and in the Armed Forces.

5. It is a useful adjunct to conventional perimetry and, since the equipment is portable, it is useful for bedside examination of the visual field.

5. It may be of value as a research instrument in the study of the "extinction" phenomenon.

384 Post Street (8).

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PRIMARY GLAUCOMA: PATHOGENESIS AND CLASSIFICATION

OTTO BARKAN, M.D.
San Francisco, California

Before the discovery of the ophthalmoscope, only acute absolute and secondary glaucoma were recognized. It was not until cupping of the nervehead was revealed that hypertension was generally realized to be the essential feature of glaucoma. Even so, Sorsby[1] states that recognition was limited to the acute and absolute varieties. Thus, in 1859, von Graefe spoke only of acute, chronic (that is, absolute) and secondary glaucoma and of "amaurosis with excavation of the optic nervehead." When Donders[2] recognized this last group as glaucoma (so-called simplex) the unifying conception was achieved.

The inclusion of "simplex" or noncongestive glaucoma under the same heading with the congestive forms (acute and absolute) created the need for a new division of the glaucomas. The division was made on a clinical basis into congestive or inflammatory and noncongestive or noninflammatory (simple) glaucoma.

This division could not be carried out clinically because, as Elschnig[3] observed, cases of each group could be converted into the other. "Inflammatory" or congestive glaucoma of acute onset could, under miotic treatment, be made to run a noncongestive simple course. Noncongestive or "simple" glaucoma could at any time, irrespective of its duration or whether its antecedent pressure had been low, high, or normal, become acutely congestive. Elschnig introduced his nomenclature of "compensated" and "incompensated," therefore, to describe the changing clinical manifestations and phases of glaucomatous eyes. He called "incompensated" glaucoma, in analogy to cardiac disorder, those cases which showed secondary congestive sequelae in the anterior part of the globe; that is, a failure of the circulatory apparatus of the eye following a rise in intraocular pressure. Elschnig's terms describe clinical phases. They do not correspond to an anatomic or causal basis any more than the previous divisions and, as before, the inclusion of cases of different pathogenesis in the same group caused confusion.

In 1923, Raeder[4] attempted an anatomic classification based on the axial depth of the anterior chamber. However, the questionable significance of the axial chamber depth and its marked variation in normal eyes and in both forms of glaucoma limited its value. Raeder's investigations, as well as the succeeding ones of Rosengren,[5] who measured the axial depth of the anterior chamber with the apparatus of Linstedt, were necessarily restricted, in the absence of gonioscopy, by inability to examine the angle of the anterior chamber.

In 1930, Rosengren,[5] in a basic statistical study, showed that eyes in which glaucoma is associated with a shallow axial anterior chamber depth are characterized by the occurrence of acute attacks in the majority of cases; however, in certain instances the glaucoma took the form of uncomplicated glaucoma simplex. He suggested that further study of the connection between axial depth of the chamber and glaucomatous increase in pressure would require investigation by another method.

Gonioscopy proved to be such a method. By means of it, it was shown by me in 1938,[6, 8, 22, 40] that the groupings into "congestive" and "noncongestive" or "simple" and into "compensated" and "incompensated" did not correspond, except in a general way, to pathologic-anatomic entities and that as a result they were often misleading.

Furthermore, measurements of the axial depth of the anterior chamber when combined with gonioscopy showed that the axial

depth is in many instances an unreliable indicator of the form of glaucoma since it often does not correspond to the changes in the angle which are the local cause of the increased pressure.[7]

Classification and nomenclature on basis of local pathogenesis

Since I first suggested that glaucoma be divided into two groups on the basis of the changes in the angle, that is, on a local pathogenic basis, all of my succeeding observations have supported this view.[7, 19, 23, 66] Other clinical characteristics, including the presence or absence of congestion, history, and axial depth of the anterior chamber were used to aid in the classification. Some further distinguishing features are being published in the present article. The groups consist of:

1. Those in which the local cause of the increased pressure is blockage or closure of the filtration angle by the root of the iris.

2. Those in which closure of the angle (iris-block) does not occur and the obstruction, either functional or organic, which is the cause of the increased pressure is presumably localized within the trabeculo-Schlemm's canal mechanism including its emissaries.

In 500 successive primary glaucomas observed in the present study 45 percent were of the narrow-angle (iris-block) variety and 55 percent open (wide) angle glaucomas.

Since, in eyes of the first group, narrowness of the entrance to the angle was a gonioscopic feature in all stages of the disease and always preceded closure of the angle and increased pressure, the term "narrow-angle" was chosen as a convenient label to indicate this form. In order to avoid possible misinterpretations the term "iris-block"[8, 22] was added as being more expressive of the underlying pathogenic concept of angle closure.

The term "wide-angle" glaucoma was originally applied to the second group in order to emphasize the fundamental difference from, and to contrast it with, the "narrow-angle" type, even though the angle might be fortuitously narrow. *The angle can be of any width.* I, therefore, suggested the term "open-angle (non-iris-block)" as being more accurate although more awkward. "Trabecular" glaucoma was also suggested as probably indicating the local cause in some eyes of this group.[8, 22]

These terms were introduced to identify and separate cases of what appeared to be two fundamentally different disorders. The classification and nomenclature received wide acceptance and the observations upon which they were formed have been confirmed by many workers.[9, 10, 11, 20] This nomenclature should not be interpreted literally to mean that the width per se of the angle influences the intraocular pressure or determines the form of glaucoma as has been implied by some authors.[12-14] The symposium on glaucoma of the American Academy of Ophthalmology and Otolaryngology[67] in 1948, which advocated the use of the terms "narrow" and "wide-angle" glaucoma encountered criticism by Duke-Elder,[12] and Magitot,[14] and others, largely, it appears, because the terms had been defined and interpreted without the qualifications originally given as to their intended meaning. One is reminded of the statement of von Graefe[15] that probably no subject in ophthalmology has given rise to more controversy than the question of the etiology and nature of glaucoma.

The significance of the grouping on the basis of the angle findings cannot be overemphasized. On this basis the subject is also kept open for further study without preconceptions while at the same time making it possible to acquire valuable information by contrasting the characteristics of the two groups.

It is conceded that the time-honored division into "congestive" and "noncongestive" or "simple" is in many cases rather closely related to the clinical picture. However, there are many cases of glaucoma in which no con-

gestive history can be elicited and which run a "simple" course uncomplicated by congestive signs but which are potentially congestive (iris-block) and, therefore, belong to the congestive group as regards the therapeutic and surgical implications (iridectomy).

When confronted by this difficulty of classification, Graefe and Donders[3] grouped the clinically noncongestive cases together, the former under the heading "amaurosis with excavation of the optic nervehead" and the latter under "glaucoma simplex." In this noncongestive group they included cases with shallow chamber which later became congestive, as well as noncongestive companion eyes in cases with acute symptoms in the first eye. According to the present classification on the basis of the local cause in the angle, namely, whether the cause is closure of the angle by the root of the iris or not, these eyes would come under the heading of narrow-angle iris-block glaucoma which responds to iridectomy whereas the other members of the "simplex" group with permanently open angles do not respond to iridectomy.

The inclusion of these noncongestive narrow-angle (iris-block) cases in the same group with the open-angle or true simple glaucomas has been the cause of much confusion. If the term "simple" glaucoma is used, I believe that in order to avoid confusion it should be qualified by "open-angle" to indicate that the angle stays open and that the increased pressure is not the result of closure of the angle by the root of the iris. The two nomenclatures could be combined in the term "simple open-angle glaucoma."

The classification and nomenclature on the basis of the local cause of the retention can be applied mutatis mutandis to the secondary glaucomas.

Present investigations

The results are presented of successive gonioscopic and other observations made over the course of many years in over 200 nonglaucomatous and 700 eyes with primary glaucoma. Measurements of the axial depth of the anterior chamber and of the corneal diameter were made in 500 eyes. A comparison of pre- and postoperative conditions in the angle and in the anterior chamber is combined and correlated with the other clinical findings.

An analysis and explanations are presented for some of the observed anatomic, mechanical, and functional changes. They are considered as links in the diencephalic neurohormonal mechanism, a function of which is the regulation of the intraocular pressure. This mechanism has been described by Thiel,[20] Magitot,[14] and others. It is referred to in the following as the central control mechanism.

Method of gonioscopy

The comparison of narrow-angle (iris-block) and open-angle glaucoma serves as an important instrument of investigation. However, changes in the angle mechanism can be adequately observed and compared only by using certain gonioscopic techniques.

The present study was based on a method of gonioscopy, which has been developed by me over the course of years,[22] using a slight variation of the Koeppe glass contact lens,[11] a focal illuminator devised for the purpose,[21] and a binocular microscope mounted by universal joints on an adjustable and movable floor stand, with the patient in the recumbent position. The ceiling suspension model can also be used. Total magnification is ×30 (50 mm. objective and ×10 oculars). As the depth of focus is short the system requires very sharp focusing and stability. By means of the focal illuminator the incident beam of light can be given any desired direction. It permits examination by reflected light and by trans- and retroillumination. Its brightness and flexibility are much greater than those of the slitlamps in use today. As a result of these features this method gives markedly greater resolving power than those

which employ a plastic lens with the patient sitting upright at the slitlamp microscope.[23, 31] Also, fine adjustments between glass, eye, and observer are more easily made.

The focal illuminator provides an optically imperfect slit (the image of the filament) but it is preferable to the optically perfect slitlamps in use today because its brilliance and flexibility are so much greater. Thus, with this method it has been possible to observe structures and details both in children and in adults (such as, to mention only one example, the surface shagreen [endothelium] covering the angle wall and parts of the iris and its crypts) which have not been reported by other methods.

Results of investigations

Corneal diameter

The average corneal diameter in *nonglaucomatous eyes* was found by Priestly Smith[25] to be 11.6 mm. In the present series the corneal diameter, measured in the horizontal meridian with a Burch eye caliper in over 150 nonglaucomatous eyes, was 11.5+ mm.

In open- (wide-) angle glaucoma (over 150 eyes) the average corneal diameter was the same as in nonglaucomatous eyes.

In narrow-angle (iris-block) glaucoma the corneal diameter was smaller than average in 80 percent (less than 11.5 mm.). It is probably permissible for purposes of comparison to regard any cornea with a horizontal diameter of less than 11.0 mm. as unusually small. This was the case in over 30 percent of narrow-angle glaucomas.

Axial depth of anterior chamber

Measurements were taken with the Ulbrich drum from the vertex of the cornea to the edge of the pupil (2.0 mm. wide) in over 500 cases of primary glaucoma. These measurements are direct readings from the drum without applying the corrective formula of Hartinger.[26] Recently these measurements were checked with the method of Linstedt[27] (apparatus of Stenstrom[28]) and found to be accurate to within 0.1 mm. They were, therefore, sufficiently accurate to be used comparatively between one another. However, since they do not represent values of the actual axial depth of the anterior chamber, they cannot be compared with measurements of this dimension obtained by other methods.

The average axial depth of the anterior chamber measured in this manner in 150 *nonglaucomatous eyes* was 2.0 mm.

In open- (wide-) angle glaucoma the axial depth of the anterior chamber showed the same variations as in nonglaucomatous eyes. It may occasionally be fortuitously shallow.

In narrow-angle (iris-block) glaucoma the axial depth of the anterior chamber was less than 1.5 mm. in 74 percent. It is probably permissible for purposes of comparison to regard any axial chamber depth under 1.5 mm. as unusually shallow. In 80 percent of these the cornea was unusually small. In the remainder the cornea was of average size.

The axial anterior chamber depth was average in 20 percent. In 75 percent of these the cornea was of average size. The latter group included cases of typical acute glaucoma in which closure of the angle by the root of the iris was proved by gonioscopy and by the effects of iridectomy to be the local cause of the increased pressure. From this it may be concluded that shallowness of the axial depth of the anterior chamber with a small cornea is a frequent but not an essential feature of narrow-angle glaucoma.

A normal axial depth combined with an average size cornea was found in nine percent of cases.

Acute glaucoma may occasionally have a normal corneal size and a normal axial depth of the anterior chamber. Nevertheless, in these cases the angle was also found narrow before and closed during the acute attack.

The terms "shallow-chamber" and "deep-chamber" glaucoma which are still occasionally used do not correspond except in a gen-

eral way to the two groups and may be misleading in as much as the axial depth is not in consistent relationship to the conditions of the angle or to the local cause of the retention.

I have observed gonioscopically through a surgical coloboma that in eyes with a small cornea the perilental space is smaller than in eyes with an average size or large cornea. This observation supports the evidence of anatomic sections cited by Priestly Smith[23] that in small eyeballs the lens may be disproportionately large and be the cause of axial shallowness of the anterior chamber. Priestly Smith found that axial shallowness could also be the result of anterior displacement of the lens.

I have measured and shown in the living subject that the progressive shallowing of the axial depth of the anterior chamber was in many cases the result of anterior displacement of the lens. Furthermore, by relating it to the increased seclusion of the pupil it was shown that anterior position of the lens besides being primary and predisposing to the pupillary block and to the development of the glaucomatous process was often the result of the glaucomatous process.[7]

Mechanical factors and phasic variations in ocular tension in narrow-angle glaucoma

If one combines the data presented in the foregoing with the known effects of axial shallowness of the anterior chamber (that is, increased seclusion, narrowing of the angle, and closure of it,[9]) it appears probable that the increased pressure in a large percentage of narrow-angle (iris-block) glaucomas is primarily of mechanical origin. Therefore, in this sense these glaucomas may be regarded as secondary to local anatomic conditions.

If the above is true, then in these cases increased phasic variations in the tension could be regarded as reactions secondary or reflex to derangement in the peripheral end-organ. This view is supported by the results of Miller's examination[17] of the diurnal variations in 25 cases of "congestive" (that is, narrow-angle iris-block) glaucoma during the interval when the angle was open, the tension normal, and there were no congestive symptoms. In 20, the diurnal variations were normal and in five only slightly exaggerated. In those in which there was obstruction the diurnal variations were definitely exaggerated.

The exaggerated diurnal variations are reduced by thalamic sedatives. This indicates that they are under the influence of the central mechanism but not that they are necessarily the result of a primary derangement of it.

Role of seclusion of the pupil in narrow-angle glaucoma

In other articles[6,7,22] factual evidence has been presented to show that seclusion of the pupil (relative pupillary block) is a major causal factor in narrow-angle (iris-block) glaucoma. It was shown that it is caused by a disproportionately large lens or by an anterior displacement of the lens. It, in turn, can produce further anterior displacement of the lens. Other causal factors may be involved. It was noted that theoretically the degree of seclusion may be influenced by vascular or neurogenic derangements which change the composition of the intraocular fluid or its rate of flow and that the latter changes may well be and probably often are secondary or reflex to obstructive changes (functional or organic) in the end-organ.

The action of peripheral iridectomy in collapsing the bombé showed that the preoperative pressure in the posterior chamber had been increased by the seclusion of the pupil. The retroplacement of the lens which followed iridectomy indicated that the pressure in the vitreous compartment had also been increased by the seclusion and by its effects on the angle and on the pressure in the posterior chamber. Evidently the pressure in the posterior chamber and in the vitreous

compartment are functionally linked.[7] Thus, peripheral iridectomy performed through a self-sealing or with a completely sealed incision, by reducing the pressure in the posterior chamber also reduces the pressure in the vitreous compartment. It may do this directly or indirectly by reflex action on the formation of fluid.

Peripheral iridectomy performed through a self-sealing or with a completely sealed incision was found consistently to relieve the seclusion, reduce the bombé, widen the angle throughout its circumference, and normalize the intraocular pressure in narrow-angle glaucoma, provided that there are no adhesions in the angle and the trabeculum which is exposed as a result of the collapse of the bombé is normally permeable. In many cases it retroplaces the lens. Thus increased seclusion of the pupil, closure of the angle, and anterior displacement of the lens constitute a vicious circle which can be interrupted in early cases and reversed.[7]

Pathogenesis of primary glaucoma

It is interesting and of advantage to combine a study of the demonstrable local anatomic changes in the peripheral end-organ with the neurohormonal point of view.

It is generally agreed that physiologically a central control mechanism participates in the regulation of intraocular pressure and flow of fluid through the eye and that the objective of the mechanism is to maintain optical function, metabolism, and fluid and vascular circulation. The globe may be regarded as the peripheral end-organ of this mechanism, possessing afferent and efferent pathways, both neural and hormonal.

Primary disturbance of the neurohormonal mechanism in any of its planes may affect the local regulating mechanism. It may do so by influencing the vascular flow, the capillary permeability, the secretory epithelium, or the composition of the fluid, resulting at first in functional and later in organic impediment to flow. The peripheral impediment, irrespective of whether functional or organic, would secondarily and reflexly again disturb the central control mechanism. Thus, once the control mechanism has been disturbed by an obstructive peripheral derangement, either functional or organic, in order to restore it to functional equilibrium it is necessary to remove the obstruction in the end-organ and re-establish flow by means of drugs or certain types of surgery which re-establish the physiologic direction of outflow (iridectomy or trabeculotomy), or to relieve the increased pressure by creating a vicarious channel of outflow (fistulizing operations), or by reducing the formation of fluid (cyclodiathermy or secretion inhibiting drugs). The complicated interplay of functions emphasizes the importance of dividing these disorders into groups on a factual basis as far as this is possible.

Thiel[20] has summarized the evidence which proves the existence of a regulating center. The factual and experimental evidence at hand for evaluating this regulating mechanism is growing.

But there is still much opportunity for analysis in the end-organ. The evidence, cited in this and previous articles, appears to show that at the present time an analysis of glaucoma can fruitfully be pursued on the basis of a demonstrable fundamental factual similarity or difference, as the case may be, in the disorders of the end-organ and that much information can be acquired by comparing them.

The common denominator in one group is closure of the angle, or iris-block with its associated changes. The common denominator in the other group is that the angle is always open.* The increased pressure is due to factors presumably located in or near the angle wall (trabeculo-Schlemm's canal mechanism including its emissaries).

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* The angle is closed by the root of the iris only occasionally in the late degenerative stage of absolute glaucoma.[6]

Pathogenesis of narrow-angle (iris-block) glaucoma

Narrow-angle glaucoma appears to be primarily a mechanical derangement in the end-organ in which narrowness of the angle results from increased seclusion of the pupil.[6–8, 29, 30] The entrance to the angle having been narrowed to a mere slit, its closure can be precipitated by mydriasis,[8, 10, 31–33] vascular engorgement, mental stress, or any derangement of the central control mechanism. The mechanical predisposition appears to be the sine qua non of iris-block (angle closure) glaucoma.

As Scheie[16] states, however, the adherents of the gonioscopic school assume that the underlying disturbances and physiopathology are largely unproved, thus leaving the subject open for further study.

Duke-Elder[12, 36] suggests that the immediate or local cause of congestive glaucoma is edema of the globe due to uveal vascular congestion and that narrowness of the angle is not causal, but is merely an incident which may affect the course of the disease.

Rosengren[34] recalls the statement of Priestly Smith that congestion of the uveal tract is a usual starting point of an acute primary glaucoma but that this explanation of the origin of acute glaucoma is obviously incomplete since feeble circulation, chill, mental strain are common troubles whereas acute glaucoma is comparatively rare; also that E. Fuchs[35] pointed out many years ago that myopic eyes, having a deep chamber, are apparently immune to acute glaucoma and concluded that "we are forced to adopt the view that the structure of the eye is an essential factor in acute glaucoma."

The evidence at the present time encourages the view that the congestion which is observed clinically and in anatomic specimens is secondary to closure of the angle and the resultant retention and increased pressure.

The primary and intrinsic derangement appears to be narrowness of the angle (the result of seclusion and bombé) which precedes it.

It is agreed that a slight congestion may precipitate closure of an already severely handicapped angle. In reviewing a large number of narrow-angle and open-angle glaucomas, one gains the impression that a greater percentage of individuals in the former group show signs of emotional or vasomotor instability. Whether the instability is an underlying causal factor or acts only as a precipitating cause in a structurally and fundamentally predisposed eye remains a question. The greater hereditary tendency in narrow-angle[8, 37] over open-angle glaucoma which is presumably on the basis of anatomic characteristics supports the latter explanation.

Tornquist[37] has recently established the importance of genetic factors in determining the axial chamber depth. Previous studies by Rosengren[34] had indicated that a shallow anterior chamber exists before the onset of glaucoma and thus is a predisposing factor. That it may also be acquired has been shown by me in the present and recent articles.*

That narrow-angle glaucoma is primarily a mechanical or structural derangement is also supported by the fact that when the bombé of the iris is collapsed and the angle widened by short-circuiting the seclusion of the pupil by mechanical means (peripheral iridectomy or iridotomy performed with a sealed incision) the pressure-raising effects of emotional disturbance or vascular engorgement are discouraged or entirely eliminated, provided always that the trabeculum is still normally permeable at the time of operation.

Haas and Scheie[72] and Chandler[29] have recently reported similar results.

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* A shallow chamber is practically never found after infancy. It is not known at what age the shallowness of the chamber which is characteristic of narrow-angle (iris-block) develops. It may be that the physiologic decrease in size of the cornea combined with the growth of the lens encourages increased seclusion with consequent secondary shallowing of the anterior chamber in genetically predisposed eyes.

In narrow-angle glaucoma the intraocular tissues show no pathologic changes except those which are secondary to the increased pressure. There is no primary dispersion of pigment as there is in open- (wide-) angle glaucoma which might indicate an underlying vascular, atrophic, or degenerative disturbance. The trabeculum is normal unless it has been damaged secondarily by contacts with the iris. The tissues in narrow-angle glaucoma, in contrast to those of open- (wide-) angle glaucoma, react to surgery like the tissues of normal nonglaucomatous eyes.

It is characteristic of narrow-angle glaucoma that before the optic nerve has been damaged by pressure it shows a very small or almost no physiologic excavation. This may be in relation to the small and fibrous eyeball. It is in marked contrast to the findings in open-angle glaucoma in which the cornea and globe and the physiologic excavation are of larger size. It may be that in the latter the disc is less resistant to increased pressure. It was also found characteristic of narrow-angle glaucoma that damage to the optic nervehead was closely correlated to the intraocular pressure. True normalization of the pressure resulted in arrest and preservation of the function of the nerve in all but late cases in which the nerve fibers and vessels had been irremediably damaged, and the nervehead was predisposed to further atrophy no matter how low the pressure was reduced.

Thus, injury to the optic nerve fibers and vessels appears, in narrow-angle glaucoma, to be primarily on a mechanical or pressure basis. In chronic cases injury to the nerve does not manifest itself until the fibers or vessels are pressed against and kinked at the scleral rim of the nervehead. Hence, an appropriate and safe mechanical procedure such as peripheral iridectomy performed with a tightly sealed incision is indicated as an early or prophylactic operation before damage has occurred to the nerve at the scleral rim, while the trabeculum is still normally permeable, the base pressure[47] has not increased, and the pathologic narrow-angle process is still reversible. Such an early or prophylactic iridectomy has proved eminently satisfactory in practical experience.[6, 8, 19, 20] Its effectiveness has recently been emphasized by Chandler[20] and by Haas and Scheie.[72]

Pathogenesis of open- (wide-) angle glaucoma

In all cases of this group the angle remained open except occasionally in the last stage of absolute glaucoma when it became closed as the result of secondary changes. The axial depth of the chamber also remained characteristically unchanged in marked contrast to the course of events in narrow-angle (iris-block) glaucoma.

Changes in the trabeculum could be made out gonioscopically in 90 percent of cases. In 65 percent there was a trabecular pigment band associated with trabecular sclerosis. In 25 percent there was no pigment band but there was trabecular sclerosis as indicated by reduced permeability of the trabeculum to direct light or to transillumination. In 10 percent the appearance of Schlemm's zone of the trabeculum was normal. The latter finding does not prove that an anomaly of the trabeculo-Schlemm's canal mechanism was not present since tonography showed increased resistance to outflow in the region of the trabeculo-Schlemm's canal mechanism also in these cases.

What the primary causal factor may be in these cases is as yet the subject of speculation. A disturbance of the neurovascular control mechanism as exemplified by increased episcleral venous pressure and suggested by exaggerated diurnal variations of pressure has been hypothecated by Duke-Elder,[13] Thomassen,[46] and others. Ascher[63] has suggested a constriction of the collector channels. Ashton's[68] observations with Neoprene injections tend to support this suggestion.

In some cases of several years' duration,

TABLE 1

CONTRAST OF OPEN- (WIDE-) AND NARROW-ANGLE GLAUCOMA

	Open- (Wide-) Angle Glaucoma (noniris-block)	Narrow-Angle Glaucoma (iris-block)
Axial anterior chamber depth	Varies as in nonglaucomatous eyes	Shallow in 80%
Corneal diameter	Varies as in nonglaucomatous eyes	Small in 74%
Size of lens	Varies as in nonglaucomatous eyes	Disproportionately large in many
Refraction	Varies as in nonglaucomatous eyes Occurs also in myopia In young adults pigment glaucoma is associated with myopia	Predisposed by hyperopia Never occurs in axial myopia
Heredity	Occasional	Frequent. Reduced chamber depth is related to genetic factors
Incidence	Age at onset: Over 50 years	Age at onset: Over 40 years
Monocular or Binocular	May be monocular for years	Predisposition is always binocular; glaucoma is usually binocular within short space of time, unless interrupted by miotics
Sex	No predisposition	More frequent in females
Pigment changes	Disturbance of pigment epithelium Dispersion of pigment in anterior segment with deposition on posterior surface of cornea and/or in Schlemm's zone of trabeculum in 65%	No disturbance of pigment epithelium or dispersion of pigment in anterior segment except secondary to congestive attacks, angle closure or surgery. Depigmentation of iris stroma in patches corresponding and secondary to areas of closed angle
Gonioscopy	Angle is open. Width of angle varies as in nonglaucomatous eyes. In 65% a trabecular pigment band of greater or lesser degree impregnates Schlemm's zone throughout its circumference. In 90% the trabeculum shows reduced permeability to light as a manifestation of more compact and impermeable structure, e.g., trabecular sclerosis	Narrow-angle; in 80% associated with shallow depth of the axial anterior chamber, seclusion of the pupil and bombé of the iris. This results in closure of the angle. In 20% the axial anterior chamber depth is normal and the angle is narrowed and becomes closed by the last fold of iris contacting the angle wall. Trabeculum is normal until angle closure occurs. Contact with root of iris results in secondary sclerosis of trabeculum and/or infiltration with pigment thus producing a "secondary" trabecular pigment band
Tonography	Increased resistance to outflow in angle wall (trabeculo-Schlemm's canal mechanism)	Normal facility of outflow until changes in trabeculum occur as result of previous contact with iris or the angle is closed
Diurnal tension	Increased variations	Normal variations when angle is open. Increased variations when part of angle is closed
Rate of flow	Normal	Increased (?) or variable (Goldmann)
Effect of miotics	Reduce tension in varying amounts, probably by means of vascular action and/or unfolding of trabeculum	Very effective, mainly by means of opening the angle; perhaps also by vascular action and/or unfolding of trabeculum
Effect of mydriatics	Slight, if any, increase of tension	Increase of tension; may induce acute attack
Effect of darkness	No effect	Increase of tension

TABLE 1—(*continued*)

	Open- (Wide-) Angle Glaucoma (noniris-block)	Narrow-Angle Glaucoma (iris-block)
Effect of water drinking	Increase of tension	No effect
Optic nerve	Preceded by average physiologic excavation. Marked glaucomatous excavation appears early relative to other signs and symptoms	Preceded by unusually small or absent physiologic excavation. Pathologic excavation occurs only when base pressure is increased with or without congestion (result of closed angle or secondary changes which reduce permeability of trabeculum)
Clinical symptoms	Generally absent; very exceptionally halos or transient blurs as result of rapid increase of tension. Usually no episodes of vascular congestion	Prodromata consisting of halos, fogs, pain. Clinical signs of vascular congestion are secondary to closure of the angle
Pathologic anatomy	Enucleation is rare since globes are without pain even in late stage (except following unsuccessful surgery or in the late degenerative stage of absolute glaucoma)	Enucleation is frequent since globes are painful. Therefore, most eyes enucleated for glaucoma are of the narrow-angle (iris-block) type
	Angle is open; trabeculum shows atrophy and sclerosis usually associated with pigment infiltration. Angle does not close except as result of secondary degenerative changes in the late stage of absolute glaucoma	In the early stage the angle is open in some areas but extremely narrow; in others it is closed. The root of the iris is reflected 180° upon itself. The posterior chamber is deep. In the late stage the angle is closed by peripheral adhesions in characteristic manner
Pathogenesis	Retension is caused by impediment to outflow in or near the angle wall (trabeculo-Schlemm's mechanism). Angle remains open (noniris-block). Axial depth of the anterior chamber does not change during the course of the disease	Retention is caused by closure of angle by root of iris (iris-block). This is the result of increased functional seclusion of pupil, bombé of iris, narrowing of angle and angle closure associated with secondary expansion of vitreous chamber and increased anterior displacement of lens. A vicious circle ensues. Retroplacement of lens follows iridectomy in some cases. Axial depth of the anterior chamber changes in the course of the disease

I have observed gonioscopically that Schlemm's zone of the trabeculum and the blood-filled Schlemm's canal were abnormally narrow. This suggested anatomic deficiency of the mechanism which may be related to reduced functional capacity.

Disturbance of the unfolding of the trabeculum may also be a factor.[41, 42] However, there is no method at the present time by which changes in permeability of the trabeculum which are the result of folding and unfolding (changing structure) can be tested.

A normal gonioscopic appearance of the angle wall was more frequent in early cases than in late. The percentage given would thus depend somewhat upon the material observed as well as upon the method used.

In the present series some deposition of pigment was found on the posterior surface of the cornea in 63 percent of open- (wide-) angle glaucomas. This was present in some cases in the early stages before the pressure had increased to a marked extent. In many cases it consisted only of several disseminated dots of pigment, in others of extremely fine gray dots sometimes in large number and dispersed over the greater part of the corneal surface. *What was striking was not*

so much their presence but the contrast to their complete absence in narrow-angle glaucoma provided that in the latter no congestive episodes had occurred nor surgery been performed. I have not found this mentioned in the literature. It suggests a disturbance in the secretory process and composition of the aqueous.

Trantas[43,44] reported abnormal pigmentation of the trabeculum in two thirds of their cases of "simple" glaucoma, and Bangerter and Goldmann[18] in 36 percent. François[45] found superficial pigmentation in the region of Schlemm's canal twice as frequently in simple glaucoma as in normal eyes or in not more than 40 percent. Sugar[10] found it in 50 percent and considers it debatable whether the pigment deposition resulted from the glaucoma or was a coincidental senile process. Kronfeld and McGarry[9] felt that excessive pigment occurs only in glaucomatous eyes.

I have found that the tissues in open-angle glaucoma in contrast to those in narrow-angle glaucoma are prone to react abnormally to surgery in that there appears to be an increased tendency to formation of adhesions and deposition of pigment. This along with the higher incidence of pigment disturbance suggests that the tissues of the eye are diseased in open- (wide-) angle glaucoma in comparison to the initially healthy tissues in narrow-angle glaucoma.

In the early stages of the disease there are usually gonioscopic signs of sclerosis of the trabeculum or obstruction of its interspaces by particulate matter. These are presumably the result of what are initially degenerative tissue changes, metabolic, neurogenic, or functional neurovascular disturbances. In some cases (20 percent) there is retention (increased pressure or decreased facility of outflow) without there being any sign of an anatomic derangement. This suggests that the disturbances to outflow in these cases may be on a functional neurovascular or secretory basis[46] or related to an as yet not demonstrable insufficiency of unfolding of the trabeculum on a neurogenic functional basis.[41]

Later, at a time when the base pressure[47] is increased, organic changes in the trabeculum are usually demonstrable by gonioscopy and can in some cases be proven surgically (trabeculotomy) to be causal factors of the increased pressure. The major role of mechanical obstruction in the angle wall in the later stage of the disease was supported in several cases in my experience by the normalization of pressure which followed removal or incision of the trabeculum by means of trabeculotomy under gonioscopic control. Postoperative gonioscopy showed in these cases that the trabeculum alone had been incised. No cleft leading to the suprachoroidal space was present.

It is as yet impossible to say what is the intimate nature of the disturbances of function which precede and result in the obstructive organic changes in the trabeculo-Schlemm's canal mechanism. Duke-Elder[13] has suggseted that the exaggerated phasic variations of ocular pressure combined with changes in pressure in the episcleral veins into which the aqueous flows indicate a primary functional vascular disturbance, that is, a disturbance of the local vascular control. Thiel[20] in extensive examinations of a large number of glaucoma patients failed to elicite characteristic or definite changes in the humoral constitution.

It is to be hoped that intensive examinations and comparison of the findings after careful segregation of cases according to the classification suggested in this and previous articles may aid in providing factual evidence as to the underlying disturbance.

In open-angle glaucoma the optic nerve often does not respond to reduction of pressure as well as it does in narrow-angle glaucoma.[9]

It is more prone to fail progressively, suggesting that in these cases there may be a noxious factor present other than the in-

creased pressure. This is suggested by the pigment disturbance which is indicative of an intraocular process of disease and is prevalent in open-angle glaucoma, whereas it is absent, except as a secondary phenomenon, in narrow-angle glaucoma. Also, the aforementioned lesser anatomic resistance of the optic nerve in this group may play a role, or the predisposing factor to optic nerve damage may only be that of greater duration of time and/or the consistency of increased pressure in open-angle glaucoma, it being more episodic in narrow-angle glaucoma.

It has been suggested that primary vascular disease continues to affect the nerve tissue irrespective of the pressure.[12, 14, 48] On the other hand, there is much clinical evidence to suggest that the vascular lesions are secondary to and the result of the increased pressure and of the resultant mechanical derangement in the optic nervehead. They may, of course, be both.

In this connection it is interesting to recall, as has already been mentioned under the heading of narrow-angle glaucoma in the foregoing, that the cornea, and presumably the eyeball in open-angle glaucoma is larger, on the average, than in narrow-angle glaucoma; the physiologic excavation preceding the glaucoma is wider and the nervehead appears less fibrous. Also, at surgery (cyclodialysis) I have been impressed by the thinness of the sclera in open-angle glaucoma as compared to that found in narrow-angle glaucoma; all of which suggests a thinner and less resistant lamina as a predisposing factor which weakens the resistance of the nervehead to pressure.

In reviewing operated cases one gains the impression that in open-angle glaucoma in which surgery has normalized the pressure before excavation of the nerve has reached the rim there is no further deterioration of function. It is difficult to prove this because open-angle glaucomas are rarely operated upon before damage to the optic nervehead has extended to the rim at some point and a corresponding Bjerrum scotoma has developed. Over a five-year period Kronfeld and McGarry[9] found preservation of function in the early stage of open- (wide-) angle glaucoma. Reese[38] and Burke[39] have made similar observations. In the early stage the occasional case of improvement of a scotoma following surgery or the instillation of miotics is also suggestive of this view.

If operation could be performed without hazard before excavation of the nervehead has extended to the scleral rim in any part of its circumference, early or prophylactic operation might well be considered advisable in open-angle glaucoma. A review of cases of open-angle glaucoma which were carried on miotics for periods of from 10 to 15 years reveals results that recall the dictum that miotics have caused more blindness in chronic glaucoma than surgery.

However, since harmlessness and a high degree of consistency can hardly be claimed for any operations for open-angle glaucoma at the present time their performance cannot be recommended at an early period in which the base pressure is not yet increased, the peak pressures are still controlled by miotics, and the nervehead does not show signs of damage. Yet in many of these cases the reduced facility of outflow when under miotics demands further remedial action.

These circumstances appear to be the reason for the modern search for an operation which may be applied early and with safety. Retrociliary diathermy is an attempt in this direction. In the light of recent experience goniotomy or trabeculotomy under gonioscopic control in selected cases[7, 22, 49] shows occasional promise of fulfilling needed requirements.

The causes of open-angle or "simple" glaucoma are presently being investigated by many workers. Goldmann[11] places the increased resistance to outflow in the trabeculum on the basis of measurements of the outflow pressure and of the rate of flow through the anterior chamber. Grant's[41]

tonographic values for the "facility of outflow" also place the obstruction in the trabeculum. Van Beuningen's[50] goniophotometric measurements of the varying degrees of permeability of the trabeculum confirm my original gonioscopic findings and support the view of a trabecular sclerosis and of reduced trabecular permeability.[6, 49, 51] Duke-Elder[70] states that recent clinical observations are tending to show that in many cases the essential feature is an inadequacy of the ocular circulation characterized by a lack of adaptive control of the small vessels, leading both to a rise of tension and to sclerosis of important tissues in the eye.

In the present series, the frequent finding of pigment and gray dust on the posterior surface of the cornea and of pigment infiltration and sclerosis of the trabeculum in open-angle and not in narrow-angle glaucoma (except in the latter as a purely secondary phenomenon following congestive attacks or surgery) is suggestive of an obstruction of the trabeculum in many of the open-angle glaucomas at the time when they come under observation. This does not necessarily imply that it is the obstruction with pigment nor the trabecular sclerosis which is the initial cause, although it may be, but that pigment infiltration and/or trabecular sclerosis are associated with impediment to outflow through the trabeculum. There is as yet no evidence to indicate what may be the cause of the pigment dispersion in open-angle glaucoma.

Normalization of pressure by trabeculotomy in several unpublished cases supports the view of mechanical obstruction within the trabeculo-Schlemm's mechanism. On the other hand, Hobbs,[52] Busacca,[53] McLean,[54] François,[46] von Fieandt,[55] and others, using different methods of gonioscopy, have failed to find evidence of trabecular sclerosis.

The vascular factor

Gonioscopic and clinical observations made in the present study suggest that external signs of vascular congestion in "incompensated" or "congestive" glaucoma (narrow-angle [iris-block] glaucoma in the congestive phase) are secondary to closure of the angle by the root of the iris. If an acute attack of glaucoma is seen in the beginning, the eye is pale and uncongested even though the pressure is already markedly increased.

I have observed many cases gonioscopically[7] in which increased pressure followed closure of the angle and reduction of tension followed opening of the angle. In these cases closure of the angle always preceded congestive symptoms and vice versa.

This clinical fact is illustrated by mydriatic glaucoma. It has been emphasized by Sugar[10] who experimentally induced a rise in pressure by closing the angle with mydriatics in a large series of narrow-angle glaucomas in the noncongestive phase. Congestive signs and symptoms followed some time after the angle closed.

Bangerter and Goldmann[18] observed a drop of pressure which was coincidental with opening of the angle by miotics. When miotics were stopped, the angle closed and the pressure rose again. The experiments of Seidel[32] on the size of the pupil, of Serr[31] with darkroom mydriasis, and the observations of Chandler[29] on the effect of the size of the pupil on the pressure in cases of narrow-angle glaucoma also support this thesis.

Elschnig[5] arrived at similar conclusions from his pathologic-anatomic studies.

The evidence cited above argues against the view, held by many, that swelling of the ciliary body and processes closes the angle by pushing the root of the iris forward. This view was originally based on the finding of swollen and turgid ciliary processes in pathologic specimens of eyes which had been enucleated in the congested and strangulated stage. These findings were not present, however, in the rare early cases which were enucleated shortly after the onset of the attack (Elschnig, Birnbacher). On the con-

trary in them the posterior chamber was spacious and there was no sign of swollen ciliary body or processes pushing the iris forward.

It appears likely, therefore, from the clinical, anatomic, and gonioscopic evidence at hand that the marked congestion and swelling of the ciliary body and processes found in the congestive phase of narrow-angle glaucoma are the result of closure of the angle rather than its cause. They recede when the angle is opened by miotics or by peripheral iridectomy. However, it is conceded that primary congestion of the ciliary body or of the uvea due to emotional and mental states may precipitate closure of an anatomically predisposed angle.

In the present series, gonioscopy has shown in many cases, even when the axial anterior chamber depths have measured the same,* that a very slight difference in the size of the pupil or in the degree of bombé (and thus of the seclusion) between the two eyes may be sufficient to close part of the angle in one eye, thereby increasing the pressure in this eye. This shows how the slightest change—only a minute fraction of a millimeter—of configuration of an already narrowed angle is sufficient to make a critical difference in closure of the angle and in producing increased pressure. The case recently observed by me,[33] showing the influence of a unilateral spastic miosis on the course of narrow-angle glaucoma, emphasizes the critical nature of a small degree of change in the region of the angle on the intraocular pressure.

Variations in rate of outflow of aqueous described by Goldmann[56] in congestive (iris-block) glaucoma and not in "simple" (non-iris-block) glaucoma may play a role. They encourage the conception of the narrow-angle mechanism described in the foregoing and the relation of congestion to it.

A case of Grant cited by Chandler[39] illustrates the influence of slight vascular congestion. During the course of tonography, while the tension was being recorded on a paper strip, an acute rise in tension of 4.0 mm. Hg in 50 seconds, coincident with flushing of the patient's face was observed. At the end of two minutes, the tension dropped to its original level as the flushing subsided. He calculated that the equivalent volume during the rising phase of 50 seconds was 9.6 cm. mm. It would appear that such a transient volume increase could be accounted for only by a temporary increase in the volume of blood within the globe. In 1930, I observed the case of a man, aged 34 years, in whom flushing of the homolateral side of the face, unpleasant smells, or hurrying at his work, were associated with increased pressure of from 10 to 20 mm. Hg, blurred vision, seeing rainbow rings and/or occipital headache.

However, these transitory congestions, which are associated with flushing of the face or with emotions are of a very minor order when compared to the vascular changes which are clinically manifest in the congestive phase of narrow-angle (iris-block) glaucoma and which are prominent in anatomic sections of eyes enucleated for pain in the late stage of this type of glaucoma.

In the clinically congestive phase the congestion appears to be largely the result of kinking of the vessels at the root of the iris. This view is supported by the anatomic findings of Elschnig,[3] by gonioscopic evidence, and by the anatomic evidence presented by two eyes of iris-block (congestive) glaucoma successfully operated by cyclodialysis.[59] It also is the result of obstruction to venous outflow caused by the increased intraocular pressure. It appears to represent a strangulation of the vascular supply of the eye as suggested by Priestly Smith.[35] Changes in the chemical constitution of the aqueous (for instance, the presence of histamine) found in this stage appear, therefore, to be the consequence of the angle closure and associated

* With the Ulbrich drum or the apparatus of Stenstrom, designed on the principle of Linstedt.

with the congestive condition and its chemical mechanism rather than to be in a causal relation.

In this article the terms "congestive" and "noncongestive" have been used to refer to phases of congestion, that is, the presence or absence of signs or symptoms of congestion (halos, fogs or external redness). Congestion appeared in all cases to follow closure of the angle. However, the correlation is not consistent in time or degree because congestive signs and symptoms depend largely upon the rate of onset of the closure, on its extent, and on the degree of responsiveness of the individual's neurovascular system. Thus, sudden closure is followed rather promptly by congestive signs or symptoms, whereas slow and gradual closure of a considerable part of the angle may not result in congestive signs at all. In a nervously predisposed individual, closure of a minor degree may cause congestion, whereas it would not do so in an individual who was not so predisposed. This difference in reaction in narrow-angle glaucoma was observed in members of the same family, who, since the axial depths of the anterior chamber were equal, were probably predisposed by similar or equal genetic factors.

Open-angle glaucoma may exceptionally exhibit congestive signs in the form of halos. They appear to be the result of rapid changes of pressure in individuals predisposed by an unstable neurovascular mechanism.

It has been suggested that the exaggerated diurnal rhythm of intraocular pressure in glaucoma is due to lack of local vascular control and that the fundamental cause of the disease is neurogenic in nature.[13] However, exaggerated phasic variations occur in narrow-angle glaucoma only when the angle is closed. Miller[17] found that in 20 out of 25 cases of narrow-angle glaucoma examined during the interval in which the angle was open and the tension normal, the phasic variations were normal. Moreover, increased variations occur in secondary glaucoma which is caused by local mechanical change. It would appear, therefore, that in primary glaucoma the exaggerated rhythm might also be caused by peripheral obstruction as it is in secondary glaucoma; that is, that peripheral block with resulting increase of tension might induce the increase in phasic variations via neurohormonal reflex action.

Kronfeld and McGarry[9] found diurnal variations difficult to demonstrate on patients with filtering operative scars. This supports the belief that local mechanical changes (angle closure, increased pressure, and so forth) are reflexly responsible for the exaggerated diurnal rhythm and argues against a primary central disturbance as being the cause. However, it is understandable that increased primary irritability of the central mechanism would manifest itself as increased responsiveness to stimuli from the periphery and thus secondarily increase the phasic variations.

It is also possible according to Goldmann[11] that the exaggerated phasic variations which are associated with increased pressure could be explained hemodynamically.

Formation of intraocular fluid

The present article stresses the mechanism and the obstructive factors involved in the throughflow and outflow of intraocular fluid. The factor of inflow or formation of intraocular fluid has received little attention because of the scarcity of factual evidence.

It is hoped that in the future with the use of recently discovered secretion-inhibiting drugs more facts may be gathered on the role of secretion and its relation to the glaucomatous process.

Clinical evidence has suggested the presence of a secretory process which responds to varying conditions in the eye and is under neurohumoral control from both eye and brain. I have been struck, for instance, in cases of air glaucoma by the excessively rapid formation of aqueous following a period of hypertension, the result of seclu-

sion and angle closure. In one case this hypersecretion lasted for one hour in spite of repeated evacuations of the anterior chamber, before the rate of secretion abated and normal tension was restored. A similar phenomenon was observed in a case of malignant glaucoma in which fluid from the vitreous compartment was repeatedly aspirated. Rapid reformation of fluid and recurring increased tension took place during the course of an hour before normal tension was restored.

Pathologic anatomy of narrow-angle (iris-block) glaucoma

Glaucomatous eyes are usually enucleated for pain and are, therefore, generally cases of narrow-angle glaucoma. They are enucleated at a late stage when the base of the iris has become adherent to the angle wall and the angle is closed by the adhesions originally described by Weber and Kniess.

Pathologic anatomy has been of little help in elucidating the pathogenesis of narrow-angle glaucoma since anatomic specimens are rarely obtained in the early stage. Other than the closed angle, anatomic sections show vascular congestion which, as indicated in the foregoing, is most likely secondary and other changes which are also the consequence of the high intraocular pressure. They are usually so marked as to submerge any primary causal changes there may have been. However, anatomic specimens do show some of the predisposing causes. Priestly Smith found the majority of enucleated eyes were relatively small with a disproportionately large lens.

A few eyes of narrow-angle glaucoma (acute congestive attack) removed shortly after the onset of the attack were described by Elschnig[3] and Birnbacher.[57] They showed an extremely narrow entrance to the angle in places where it was still open and a configuration in the angle region which Elschnig regarded as characteristic of all cases of "incompensated" (narrow-angle or iris-block) glaucoma, namely, a rotation of the ciliary body around its axis. The iris first proceeded outward along the anterior surface of the ciliary body and then bent backward in an arc so that it formed a reduplication upon itself. There was no sign of the ciliary processes pushing the iris forward. The posterior chamber was spacious. Elschnig postulated a difference in pressure between posterior and anterior chambers in order to explain these changes and implied that they were primary.

These anatomic findings support my gonioscopic observations and surgical experiences which indicate that in narrow-angle glaucoma increased seclusion, bombé, and closure of the angle precede and are the cause of the increased intraocular pressure and of the ensuing vascular congestion.[7,10]

Pathologic anatomy of open- (wide-) angle glaucoma

Open-angle glaucomas rarely require enucleation except following the complications of unsuccessful surgery or in the late degenerative stage of absolute glaucoma. In both of these secondary changes predominate and all traces of the primary changes have been submerged. Thus, the evidence of pathologic anatomy has been of little help in deciding the question of the primary cause of the increased pressure in open-angle glaucoma.

Histopathologic changes such as the cuticular membrane covering the angle and the associated matting of the trabeculum found by Reese[65] in late cases of "deep chamber" glaucoma with advanced corneal involvement were no doubt the cause of the increased pressure at the time. However, they would appear to have been associated with and the consequence of the glaucomatous process rather than the initial cause of the increased pressure. Elschnig[3] regarded the formation of a hyaline membrane over the angle as the result of a secondary degenerative process.

Sclerosis of the trabeculum and signs of trabecular block in the presence of an open angle have been reported by De Vries,[58] Sarti,[59] and others in anatomic sections of late cases of open-angle glaucoma. They found atrophy of the trabeculae, sparsity of endothelial cells, and matting of the trabeculum.

Most of the anatomic specimens of eyes reported in the literature as early cases of "chronic simple" (noncongestive) glaucoma showing sclerosis of the trabeculum and infiltration with pigment, Levinsohn,[60] Verhoeff,[61] and others, showed peripheral adhesions in the angle. This indicates that they were really cases of narrow-angle (iris-block) glaucoma which had run a clinically noncongestive (simple) course. In them the sclerosis and matting of the trabeculum was the result of peripheral adhesions; the impregnation of the trabeculum with pigment was the result of pigment having migrated from the stroma of the iris while it was adherent to the trabeculum. In such cases when the adhesions have been released the pigment is seen on gonioscopic examination in the form of a secondary pigment band.[7, 10] The reduced permeability of the trabeculum is a result of the previous contact between iris and angle well. It manifests itself clinically by the lessened effectiveness of miotics and of iridectomy and in a few cases by the good results of trabeculotomy.

I have been able to find only one report in the literature of an anatomic specimen of indubitable primary open-angle, noniris-block or true simple glaucoma in the early uncomplicated stage, giving a detailed description of the trabeculum, that of Rönne.[62] The anatomic findings in this case because of their great significance are given herewith in literal translation:

Case Report. Male, 58 years old. Myopia −6.0. Fundus showed optic excavation to the rim. The visual fields were contracted. There had never been halos or fogs. In 1906 iridectomy was performed on the right eye. In 1911 visual acuity was worse. There were no other symptoms.

Vision was: Right, 2/24; left, 6/9.

Tension was: Right, 45 mm. Hg; left, 40 mm. Hg (Schiøtz) without miotics. After pilocarpine it was: Right, 31 mm. Hg; left 24 mm. Hg.

The patient died of uremia following prostatic hypertrophy.

Anatomic examination of the globes showed: Left eye—anterior chamber deep rather than shallow, iris stroma normal. Chamber angle wide open. The outermost portion of Fontana's spaces (trabecular interspaces) was thickened into a compact plate of sclerotic connective tissue which separated the chamber from the deeper portions of the ligamentum pectinatum (trabeculum); the latter had retained their spongy structure to a much higher degree and were more cellular than the more superficial fibrotic layer. The layers of the trabeculae and open interspaces continued up to Schlemm's canal which was essentially normal with wide lumen and not containing any red cells. The sclerosis of the superficial layers had not progressed so far as to make filtration impossible. On the contrary, in many sections more or less prominent remains of normal trabeculum were present. But the difference between the superficial and deep portions of the trabeculum was so striking and the remaining lymph interspaces were so few that this condition, in my opinion, was without a doubt pathologic. In addition, in this whole portion up to Schlemm's canal there was a marked collection of pigment and pigment cells. There were no collections of cells which would indicate an inflammatory process. Besides this sclerotic change there was no change of gross or anatomic nature.

The pigment epithelium of the retina and of the ciliary body showed no abnormalities.

Right eye. The anterior segment was the same as in the left eye except for the coloboma and corneal scar 1.8 mm. from the angle.

The findings of pathologic anatomy in open-angle or "simple" glaucoma, although few in number, are consistent with the result of gonioscopy in the present series which indicated some obstruction in the trabeculum in almost 90 percent of cases. In a little over 10 percent of early cases the trabeculum presented a completely normal permeable appearance. But, even in these there must have been impediment in the trabeculo-Schlemm's mechanism (which includes the aqueous outflow channels) since the resistance to outflow and the outflow pressure have been found increased[11] and the facility of outflow has been found decreased and been localized in this region in all cases of this type.[40] To have escaped gonioscopic detection the impediment must have been either proximal to

or within the trabecular system in the form of a functional change in the aqueous or a reduced unfolding of the trabeculum[41, 42] or situated centripetal to the trabeculo-Schlemm's canal mechanism. Ascher[63] suggested, on theoretical grounds, an impediment in the collector channels centripetal to Schlemm's canal. Friedenwald[64] suggested a theory of an osmotic drag due to plasma in Schlemm's canal, a derangement of which might increase pressure. This was contradicted by Goldmann[11] who injected fluorescein into the circulation and found that it appeared in the episcleral veins but not in the aqueous veins. Thomassen[46] has suggested that the pressure in the aqueous veins and the outflow of aqueous is controlled by the pressure within the episcleral veins. Evidence has been presented by Goldmann[11] against this theory.

There are undoubtedly several or many causal factors at work in the group of the open- (wide-) angle glaucomas. It would be premature to attempt to subdivide this group according to pathogenic factors. Nevertheless, it is tempting to think of one subdivision of open-angle glaucoma as "trabecular glaucoma"[18, 22] because of a presumable impediment either functional or organic in the trabecular mechanism.

Summary

1. The development of the classification and nomenclature of primary glaucoma in the adult is discussed.

2. The results of a study of over 500 cases of primary glaucoma are given; 45 percent were of the narrow-angle (iris-block) and 55 percent of the open- (wide-) angle variety.

3. My method of gonioscopy is described and evaluated.

4. Measurements of the corneal diameter and axial depth of the anterior chamber in open-angle and narrow-angle glaucoma are given.

5. In open-angle glaucoma the corneal diameter and axial depth of the anterior chamber measured with the Ulbrich drum and the apparatus of Stenstrom on the principle of Linstedt showed the same variations as in nonglaucomatous eyes.

6. In narrow-angle (iris-block) glaucoma the corneal diameter was smaller than average in 80 percent and unusually small in over 30 percent. The axial depth of the anterior chamber was less than average in 80 percent and unusually small in 74 percent.

7. Shallowness of the axial depth of the anterior chamber with a small cornea is a frequent but not an essential feature of narrow-angle (iris-block) glaucoma.

8. The pathogenesis of narrow-angle (iris-block) glaucoma is considered from the mechanical and neurohormonal points of view.

The reciprocal relationship between changing position of the lens, pupillary block, and closure of the angle is noted. An explanation is offered for the vicious circle.

A theory to explain the chain of events in narrow-angle glaucoma is presented.

9. The pathogenesis of open-angle (non-iris-block or simple) glaucoma is considered.

10. Characteristic features of the two main types of primary glaucoma are presented and contrasted. Deductions are made from the comparison.

11. The different structure of the optic nerve and its different resistance to increased intraocular pressure in the two types is stressed.

12. Pigment deposits are frequent in the anterior segment in open- (wide-) angle glaucoma in contrast to their absence in narrow-angle glaucoma, provided that in the latter no congestive episodes have occurred nor surgery been performed.

13. The role of the vascular factor in glaucoma is discussed. Congestion is considered to be largely the result of and reaction to obstruction either functional or organic in the end-organ.

14. It is suggested that the exaggerated diurnal rhythm may represent a reaction to

peripheral obstruction, organic or functional, in the globe.

15. Clinical evidence is presented suggesting the presence of a secretory process which reacts to obstruction and retention. It is under local and central neurohumoral control.

16. The pathologic anatomy of the two types is reviewed and contrasted.

17. The main classification into narrow-angle glaucoma (iris-block or congestive glaucoma) and open- (wide-) angle glaucoma (noniris-block, "noncongestive" or "chronic simple") glaucoma, suggested in this and other articles, appears to be justified on the basis of the given facts and to be a useful device in the diagnosis, prognosis, treatment, and investigation of the pathogenesis of primary glaucoma. It may be applied with qualifications, to secondary glaucoma.

490 Post Street (2)

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STABILITY OF AQUEOUS SOLUTIONS COMMONLY EMPLOYED IN THE TREATMENT OF PRIMARY GLAUCOMA*

PART III. EXPERIMENTAL PROCEDURE AND FINDINGS: SUMMARY AND CONCLUSIONS

W. HOWARD MORRISON, M.D.
Omaha, Nebraska

EXPERIMENTAL PROCEDURE

An attempt has been made to determine the stability of aqueous solutions of each of the following drugs: pilocarpine hydrochloride, physostigmine sulfate, neostigmine bromide, methacholine chloride, and carbachol chloride.

Di-isopropyl fluorophosphate was not included because of its property of forming hydrofluoric acid with water.[27†] Furfuryl trimethyl ammonium iodide (furmethide iodide) has been withdrawn from the market because of local inflammatory reactions, particularly that of dacryostenosis, encountered in certain glaucoma patients.[42, 98]

The miotic drugs were placed in vehicles in which they are commonly prescribed. Where it was possible a buffer, wetting agent, and preservative were included in the compound. No attempt was made to adjust the osmotic pressure of the solutions.

Pilocarpine was placed in Hind and Goyan's sodium acid phosphate and disodium phosphate buffer.[63] Benzalkonium chloride was added as a wetting and sterilizing agent. Neither the nitrate of pilocarpine nor the Gifford buffer could be used because of their incompatibility with benzalkonium chloride.

Solutions of 1.0-percent pilocarpine were compounded as suggested by Skolaut.[100]

Pilocarpine hydrochloride	0.30 gm.
Sodium acid phosphate, anhydrous	0.12 gm.
Disodium phosphate, anhydrous	0.14 gm.
Sodium chloride	0.07 gm.
Benzalkonium chloride sol. 1:5,000 q.s.	30.00 cc.

This pilocarpine solution has a pH near 6.8, which reduces its irritating quality and increases its stability at the sacrifice of some physiologic activity.[100]

Physostigmine sulfate (the salicylate is incompatible with benzalkonium chloride)

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† References for the entire paper were published with Part I and appear on pages 397-400 of the March, 1954, issue.

was placed in a solution containing boric acid, sodium bisulfite, and benzalkonium chloride. It has been determined that, if a solution of physostigmine has a pH of 6.0 to 7.0, a relatively high concentration of extremely irritating free base is formed. If, however, the pH is reduced to 5.0 or lower, the irritating sensation is largely eliminated and the drug's stability is increased without sacrificing an appreciable amount of physiologic activity.[63]

The phosphate buffer of Hind and Goyan has a pH near 6.8 and therefore should not be used. Boric acid is ideal when used to retard the rapid neutralization of a solution.[63] This weakly dissociated acid maintains the pH near 5.0.[63]

The addition of an antioxidant such as sodium bisulfite prevents physostigmine from breaking down to form a pink, irritating solution. Skolaut[100] suggested the following proportions to make a 0.25-percent solution of physostigmine:

Physostigmine sulfate	0.075	gm.
Boric acid	0.39	gm.
Sodium bisulfite	0.03	gm.
Benzalkonium chloride sol. 1:5,000 q.s.	30.00	cc.

Neostigmine bromide, 5.0 percent, was used in 1.0-percent boric-acid solution as supplied by the manufacturer.[87]

Carbachol chloride, because of its poor penetrating power, was administered with a highly surface-active (wetting) agent. A 1.5-percent solution in 1:3,000 benzalkonium chloride, as advocated by O'Brien and Swan,[84] was employed.

Methacholine chloride likewise penetrates poorly and was, therefore, also placed in 1:3,000 benzalkonium chloride to make a 20-percent solution.

The miotic solutions whose stability was to be determined were placed inside one-ounce emerald-green dropper dispensing bottles, which in turn were put in thin cardboard boxes. In this manner the solutions were stored for from three to nine months at room temperature. No attempt was made to open and agitate the contents of the bottles periodically. The pH of the solutions was ascertained with a Bechman pH meter at the time of compounding and every one to three months thereafter. Freshly prepared solutions were frozen and then permitted to return to room temperature, and other like fresh solutions were heated to 95°C. for one hour. The physiologic activity of the stored, frozen, and heated solutions was then determined.

Methods of determining drug activity

Two means were employed for testing the physiologic activity of miotic solutions:

1. Their effect on the pupils of normal young human eyes.

2. Their effect on the isolated iris sphincter of the albino rabbit.

Effect of miotics on the pupils of normal human eyes.

The size of the pupil and its response to miotics were recorded photographically. Ten female student nurses were selected, varying in age from 18 to 22 years and each having normal eyes except for refractive errors of low degree. Five of the nurses had blue irises and five had brown irises.

A room having constant indirect artificial light was used and a camera with a synchronized shutter photoflood attachment was employed. The iris and pupil were sharply focused while the photofloods were at low illumination, and the subject's gaze was directed at a distant object in line with the lens of the camera. A millimeter rule was held just below and parallel with the lower lid border. The shutter was snapped, causing the photofloods to flash to an increased brilliance and taking the picture well within the latent period of the pupil response.

A control photograph was taken of the left eye of each of the 10 subjects. One minim of the miotic to be evaluated was then dropped directly on the cornea of the left eye of each of the nurses. Four of the blue-eyed nurses received the freshly prepared miotic and four

of the brown-eyed received the miotic which had been stored. The remaining blue-eyed and brown-eyed individuals received one minim of the fresh drug as a control for the later use of heated and of frozen solutions.

The iris and pupil of the left eye of each of the subjects were then photographed at intervals of one-half hour, one hour, two hours, three hours, six hours, and 24 hours. After 48 hours, the entire procedure was repeated, with four blue-eyed nurses receiving the stored miotic and four brown-eyed the fresh solution. The remaining blue-eyed nurse and brown-eyed nurse received a solution that had been heated or frozen.

The solutions used were pilocarpine hydrochloride, 1.0 percent, physostigmine sulfate, 0.25 percent, neostigmine, 5.0 percent, and carbachol chloride, 1.5 percent. Methacholine chloride, 20 percent, failed to affect the normal pupil and its action could not, therefore, be recorded. The solutions were prepared as previously described. The heated solutions had been warmed to 95°C. for one hour and then allowed to cool to room temperature. This supposedly simulated the effect on the solutions of 30 days' time.[63]

The size of the pupil was determined by caliper measurement translated into millimeters from the coincidentally photographed rule. The determinations were accurate to 0.5 mm. A few measurements were unobtainable, because occasionally a nurse was unable to return at the proper interval for photography.

The effect of miotics on the isolated iris sphincter.

Young albino rabbits were killed by injecting air into the heart. Their eyes were immediately enucleated and placed in cold-oxygenated modified mammalian Ringer's solution having a pH of 7.4 and refrigerated at 10°C. for from five to 27 hours. An eye was then selected and, while still submerged in the cold-oxygenated Ringer's solution, the iris sphincter was removed intact by a technique modified from that described by Poos.[86] The eye was grasped and held with gauze while a small incision was made with a cataract knife into the anterior chamber at the limbus. The incision was continued with scissors until the entire cornea had been removed.

An iridotomy with scissors was performed just peripheral to the sphincter border at the 6- and 12-o'clock positions. A 5-0 white silk suture on a straight needle was passed through one of the iridotomies and tied. Care was taken not to cinch down the first tie of the knot. Another suture was passed through the opposite iridotomy and tied.

An incision to remove the sphincter was started at one of the iridotomies and was easily completed with scissors if the still intact lens and the remainder of the iris were used as support for the sphincter as it was being freed.

The intact iris sphincter was suspended in a perfusion chamber containing constantly oxygenated modified mammalian Ringer's solution (fig. 1). The volume was maintained at 20 cc. by means of continuous suction and the temperature was checked with a small centigrade thermometer.

Test solutions of miotics were injected through a No. 20 spinal-fluid needle which was long enough to reach the bottom of the chamber. A short length of flexible rubber tubing was attached to the large end of the needle so that movement in introducing the solution could not be transmitted to the apparatus.

The perfusion chamber containing the iris was kept at 20°C. by being placed in a constant temperature bath. Measured amounts of oxygenated modified mammalian Ringer's solution from a 25-cc. automatic buret were cooled to 20°C., being run through a coil in the bottom of the constant temperature bath before entering the perfusion chamber.

The suture extending from the superior

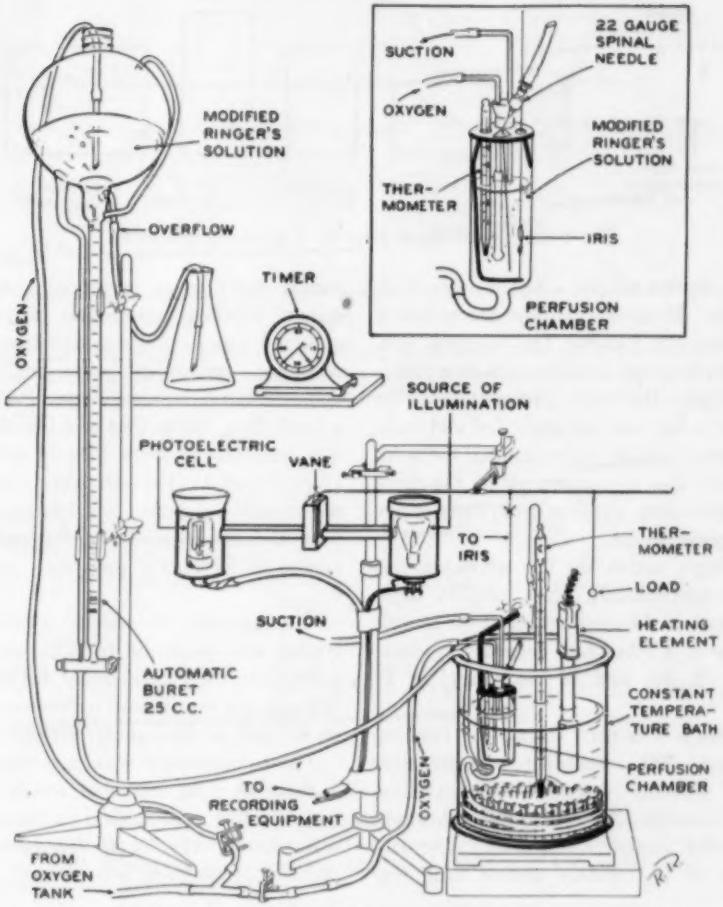

Fig. 1 (Morrison). Perfusion apparatus and transducer.

margin of the iris sphincter was tied to a hook attached three cm. from the center of a horizontally suspended aluminum rod. A thin aluminum vane painted black was attached to one end of the horizontal rod. This vane was housed in such a manner that it interrupted an intense light directed to a photo-electric cell through a 15 by 15 mm. square brass tube.

The output of the photo-electric cell was amplified and fed to two cathode ray tubes, one in a monitor oscilloscope so that muscle activity could be directly viewed and the second in an oscillograph, recording on bromide paper (fig. 2). At the opposite end of the horizontal rod was suspended a 0.361 gm. weight which could be slid along the rod, thus varying the load that could be put on the suspended iris. The suspended iris was allowed 30 minutes to recover from manipulation and to adjust to the load before being subjected to stimulation.

The miotic solutions were freshly prepared in varying dilutions from the solutions to be tested. Computation of the dilution was based on the injection of 1.0 cc. of

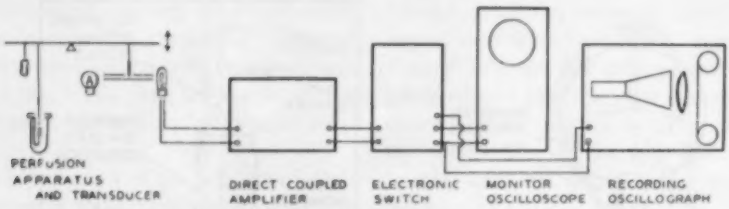

Fig. 2 (Morrison). Block diagram of recording equipment.

the drug solution to give a final known dilution in the 20 cc. of solution immediately surrounding the muscle. The solution containing the drug was introduced with a tuberculin syringe. One cubic centimeter was injected and a like amount aspirated and reinjected three times in order to mix the solution rapidly and to remove all of the more concentrated drug solution from the syringe tip and spinal needle.

The drug's action on the sphincter was recorded continuously from one to three minutes and, if the contraction of the muscle was delayed, a 10-second record was made at 5, 7, 10, 12, and, if necessary, at 15 minutes.

The muscle was then relaxed by running oxygenated 20°C. modified mammalian Ringer's solution through the perfusion chamber. Usually 125 cc. of solution brought about prompt complete relaxation. The concentration of the freshly diluted drug was varied until upon injection a slow, consistent, marked contraction of the muscle occurred. Upon obtaining satisfactory muscle response, recordings were made of the sphincter's reaction to identical dilutions of a fresh drug and to that of a like drug which had been stored from four to nine months (figs. 3 and 4). Records were also made of the muscle's response to solutions which had been frozen (figs. 5 and 6) and to those heated to 95°C. for one hour (figs. 7 and 8).

The amount of muscle contraction recorded was measured in millimeters, recognizing that the measurement did not actually indicate an equivalent millimeter response on the part of the muscle.

The same muscle was never subjected to a different drug than that which was being tested unless near the end of the experiment information other than that related to the stability of the drug was desired.

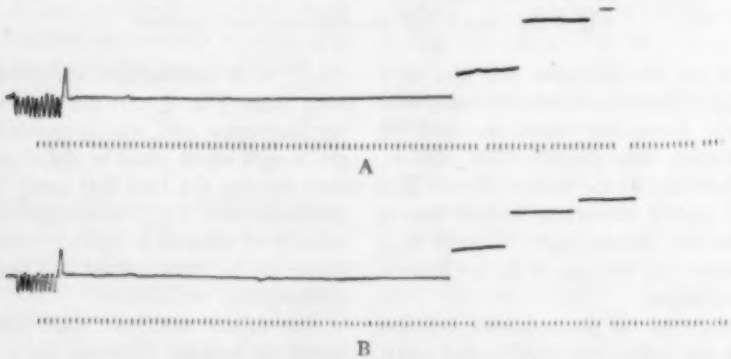

Fig. 3 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter at one, three, five, and seven minutes. (A) Induced by fresh 1:2 million carbachol chloride. (B) Induced by nine-months-old 1:2 million carbachol chloride. (Spikes indicate experiment number.)

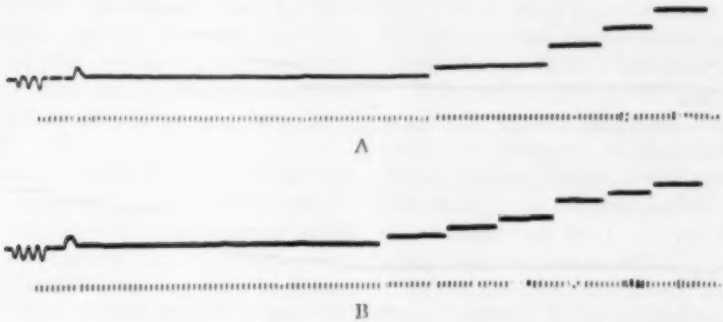

Fig. 4 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter. (A) At one, three, five, seven, and 10 minutes, as induced by fresh 1:500 thousand methacholine chloride. (B) At one, three, five, seven, 10, 12, and 15 minutes, as induced by 1:500 thousand six-months-old methacholine chloride.

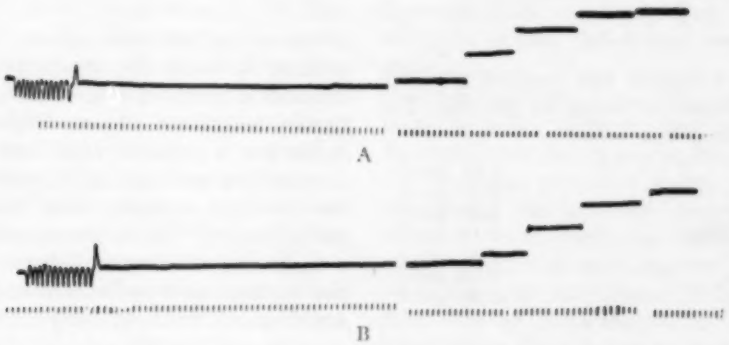

Fig. 5 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter. (A) At one, three, five, seven, 10, and 12 minutes, as induced by fresh 1:2 million carbachol chloride which had not been frozen. (B) At one, three, five, seven, 10, and 12 minutes, as induced by fresh 1:2 million carbachol chloride which had been frozen.

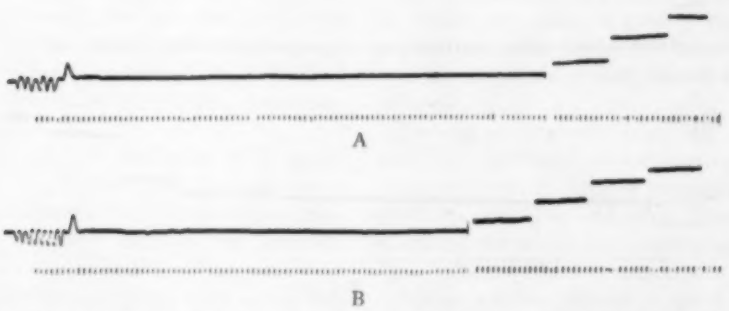

Fig. 6 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter. (A) At one, three, five, and seven minutes, as induced by fresh 1:2 million methacholine chloride which had not been frozen. (B) At one, three, five, seven, and 10 minutes, as induced by fresh 1:2 million methacholine chloride which had been frozen.

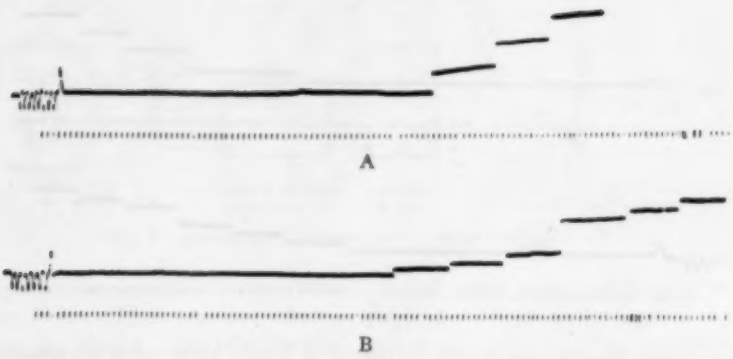

Fig. 7 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter. (A) At one, three, five, and seven minutes, as induced by fresh unheated 1:2 million carbachol chloride. (B) At one, three, five, seven, 10, 12, and 15 minutes as induced by fresh 1:2 million carbachol chloride which had been heated to 95°C. for one hour.

Comment

Ringer's solution was modified to match human plasma as nearly as possible. The formula used is as follows:

NaCl	7.00 gm./l.
KCl	0.30 gm./l.
$CaCl_2$	0.22 gm./l.
$MgSO_4 \cdot 7H_2O$	0.313 gm./l.
$NaHCO_3$	2.20 gm./l.
KH_2PO_4	0.136 gm./l.
Dextrose	2.00 gm./l.

pH 7.4

The isolated iris sphincter was viable for at least 72 hours. Its sensitivity to drug stimulation increased as the time after enucleation lengthened. It was frequently observed that a sphincter which had been denervated for as long as 72 hours was so sensitive that a suitable drug dilution was difficult to find. This increased sensitivity of a denervated structure to a chemical mediation has been noted by Cannon[21] and by Shen and Cannon,[99] and is probably accounted for

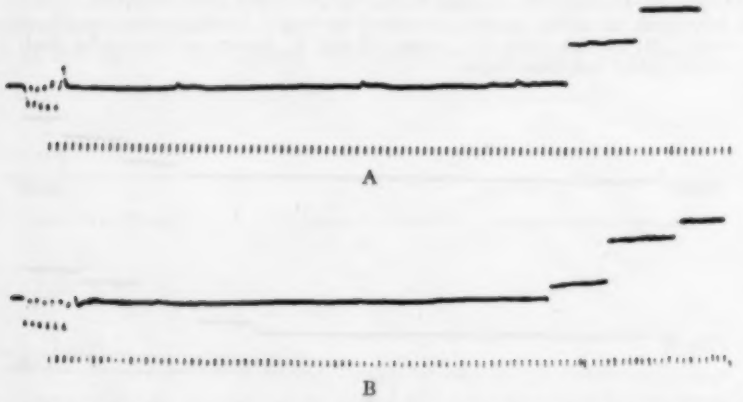

Fig. 8 (Morrison). Oscillograms, showing contraction of isolated albino rabbit iris sphincter. (A) At one, three, and five minutes, as induced by fresh unheated 1:200 thousand methacholine chloride. (B) At one, three, five, and seven minutes, as induced by fresh 1:200 thousand methacholine chloride which had been heated to 95°C. for one hour.

by de Roetth's observation[92] that cholinesterase activity rapidly decreased after iris denervation. The iris muscle cells are therefore poorly protected by cholinesterase and any acetylcholinelike drug acts with little inhibition.

Preliminary experimentation revealed that the action of the isolated albino iris sphincter was extremely erratic if the temperature of the solution bathing the iris muscle was maintained at 33°C., as suggested by Heath and Sachs.[60] It was determined by trial and error that the best temperature for constant, reproducible albino rabbit iris sphincter action was 20°C.

The muscle began to lose its reaction to a given dilution of drug after being stimulated about 10 times. Occasionally the muscle would increase in contraction after uniformly reacting a number of times to identical drug dilutions. This was believed due to an accumulation of the drug as a result of insufficient washing between experiments.

It was observed, as also by Sachs and Heath,[93] that it was unsatisfactory to reduce the drug concentration until a minimal reaction occurred. A minimal iris contraction was too inconsistent and difficult to record to be of value. An attempt was made to evaluate statistically the time of initial muscle contraction, height of contraction at various intervals, and the rapidity of relaxation. This proved to be impracticable, and so only the maximal amount of muscle contraction was used as a means of determining drug activity. This has been termed by Sachs and Heath[93] the maximally effective concentration or "M.E.C."

The isolated iris sphincter was bathed separately with Ringer's solution, Hind and Goyan phosphate buffer, benzalkonium chloride, and the boric acid sodium bisulfite combination used with physostigmine. None of these solutions produced even a minimal stimulation.

Pilocarpine failed to stimulate either the pigmented or the nonpigmented iris sphincter of the rabbit. This agrees with Guyton's[87] observation that, in the rabbit, pilocarpine produces a relatively slight miosis.

Sachs and Heath[94] found that physostigmine was capable of stimulating the isolated iris sphincter of the albino rabbit. Riker, Wescoe, Cattell, and Shorr[89] believe that neostigmine not only inhibits the action of cholinesterase but is capable of directly stimulating the muscle cell.

It was therefore anticipated that the activity produced by physostigmine and possibly that caused by neostigmine could be used in experiments with the isolated iris sphincter of the rabbit as a means of determining the stability of these two solutions. Repeated trials with each drug, however, failed to produce satisfactory isolated iris response.

The failure with physostigmine may have been due to our using the drug initially in the experiment and never employing a choline derivative except as a final means of determining that the muscle was viable. An occasional initial contraction of the muscle when bathed with physostigmine was believed due to the inhibition of cholinesterase, thus allowing the activation of traces of acetylcholine. That this is so, is borne out by the failure to obtain even the slightest response to consecutive applications of physostigmine.

It was necessary, as a means of controlling the study, to record a greater number of contractions with the freshly prepared solutions than with the stored, heated, or frozen solutions.

Certain oscillograms (figs. 4B, 6B, 7B, and 8B) revealed slower contractions than those produced by freshly prepared miotics (figs. 4A, 6A, 7A, and 8A). This finding was not, however, of frequent enough occurrence to be used in a statistical analysis.

Albino rabbits showed a great deal of individual variation in iris response. The reaction of the isolated sphincter to stimulation was influenced by the size and age of

TABLE 1

CHANGE IN pH

Drug	No. of Solutions	Months Observed	Initial pH		Change in pH	
			Average	Range	Average	Range
Pilocarpine hydrochloride	6	3 to 9	6.36	6.19 to 6.70	−0.232	−0.06 to 0.61 −
Physostigmine sulfate	5	3 to 11	3.32	2.92 to 3.68	−0.064	+0.40 to −0.68
Neostigmine bromide	6	1 to 11	5.84	5.71 to 5.96	−0.116	−0.05 to −0.21
Carbachol chloride	6	1 to 8	7.13	6.8 to 7.4	−0.238	−0.01 to −0.38
Methacholine chloride	7	1 to 10	4.55	3.21 to 5.30	−1.02	−0.18 to −1.41

* = decrease of pH. + = increase of pH.

the rabbit, presence of grossly visible blood-filled vessels, and the length of time the eye remained at 10°C. following enucleation. It was therefore necessary to determine for each iris the dilution of drug required to produce a maximal contraction which could be rapidly and completely relaxed by washing the muscle with modified Ringer's solution.

FINDINGS

CHANGE IN pH *(table 1)*

Pilocarpine hydrochloride, 1.0 percent in 1:5,000 benzalkonium chloride, sodium acid phosphate, and disodium phosphate had an average pH of 6.36, with a range of 6.19 to 6.70 pH units. Over a period of three to nine months all six solutions became more acid, the pH changing by an average of 0.232 units, with a range of 0.06 to 0.61 pH units.

Physostigmine sulfate, 0.25 percent, in 1:5,000 benzalkonium chloride, boric acid, and sodium bisulfite had an average pH of 3.32 with a range of 2.92 to 3.68 pH units. Over a period of three to 11 months two solutions became more alkaline, one by 0.18 and the other by 0.40 pH units. The remaining three solutions became more acid by 0.06, 0.16, and 0.68 pH units, respectively.

Neostigmine bromide, 5.0 percent, in boric acid had an average pH of 5.84 with a range of 5.71 to 5.96 pH units. Over a period of one to 11 months, all six solutions became more acid, the pH changing by an average of 0.116 units, with a range of 0.05 to 0.21 pH units.

Carbachol chloride, 1.5 percent, in 1:3,000 benzalkonium chloride had an average pH of 7.13, with a range of 6.8 to 7.4 pH units. Over a period of one to eight months all six solutions became more acid, the pH changing by an average of 0.238 units, with a range of 0.01 to 0.38 pH units.

Methacholine chloride, 20 percent, in

TABLE 2

PUPILLARY RESPONSE: THE PROPORTION OF PUPILS SHOWING A RESPONSE GREATER BY ONE MM. OR MORE WITH FRESH THAN WITH OLD SOLUTION*

Drug	Hours Elapsed After Instillation					
	0.5	1.0	2.0	3.0	6.0	24.0
Pilocarpine hydrochloride	3 of 8	1 of 8	2 of 8	3 of 8	None	None
Physostigmine sulfate	2 of 8	1 of 8	1 of 8	1 of 8	2 of 8	2 to 8
Neostigmine bromide	None	None	None	None	None	None
Carbachol chloride	1 of 8	2 of 8	1 of 8	1 of 8	2 of 8	None

* Eight to 10 months.

1:3,000 benzalkonium chloride had an average pH of 4.55, with a range of 3.21 to 5.30 pH units. Over a period of one to 10 months all seven solutions became more acid, the pH changing by an average of 1.02 units, with a range of 0.18 to 1.41 pH units.

PUPILLARY RESPONSE *(table 2)*

Pilocarpine hydrochloride, 1.0 percent. At one-half hour three of the eight pupils photographed showed a one-mm.* or greater contraction with the freshly prepared pilocarpine than they did with a like eight months' old solution. The greatest difference in contraction produced by the two solutions was 1.2 mm.

At one hour one of the eight pupils showed a one-mm. or more contraction with the freshly prepared drug than with the old. The difference was 1.8 mm.

At two hours two of the pupils showed a one-mm. or greater contraction with the fresh drug than with the old. The greatest difference produced by the two solutions was 1.8 mm.

At three hours three of the pupils showed a one-mm. or greater contraction with the new than with the old drug. The greatest difference was 1.8 mm.

At six hours none of the pupils showed a difference of one mm.

The pupils of all of the eight volunteers had returned to normal in 24 hours.

There was no difference in the response to miotics of the blue and brown irises.

There was less than a 0.5-mm. difference in pupillary diameter when the freshly prepared pilocarpine was compared to the like solution which had been frozen.

A maximum difference in pupillary size of 0.6 mm. occurred when a heated solution of pilocarpine was compared to a freshly prepared unheated solution.

Physostigmine sulfate, 0.25 percent. At one-half hour, two of the eight pupils showed a one-mm. or greater contraction with the freshly prepared physostigmine than they did with a like 10 months' old solution. The greatest difference in contraction produced by the two solutions was one mm.

At one hour one of the eight pupils photographed showed a one-mm. or greater contraction with the fresh drug than with the old. The difference was one mm.

At two hours one of the eight pupils showed a greater than one-mm. contraction with the old solution. The greatest difference in contraction produced by the two solutions was one mm.

At three hours one of the eight pupils showed a greater than one-mm. contraction and this one-mm. difference was with the freshly prepared solution.

At six hours three of the eight pupils showed a greater than one-mm. contraction. This occurred in one with the old solution and in two with the fresh solution. The greatest difference in contraction between the solutions was 1.5 mm.

At 24 hours two of the pupils were still contracted by the fresh solution, one by two mm. and the other by 2.8 mm. more than by a similar old solution. The pupils of the eyes which had been subjected to the old solution had all returned to normal size in 24 hours.

The response to miotics was found not to be related to the amount of iris pigmentation.

There was a difference in sphincter contraction of 0.8 mm. or less when a freshly prepared solution was compared to a like frozen solution.

A fresh solution of physostigmine compared in pupillary response to a heated solution produced a one-mm. greater contraction at six hours. The pupils showed less than one-mm. difference in contraction at all other intervals.

Neostigmine bromide, 5 percent. A fresh solution of neostigmine and a like nine months' old solution were used and not one

*A difference in pupil size of one mm., as induced by comparative solutions, was deemed significant for purposes of this study.

of the eight individuals after one-half hour, one hour, two, three, and six hours showed a pupillary contraction of one mm. or more. There was no difference in the response of the blue and brown irises.

There was not at any interval as much as 0.5 mm. difference in pupillary size when the response produced by a freshly prepared solution of neostigmine was compared with that of a like solution which had been frozen, or one which had been heated to 95°C. for one hour.

Carbachol chloride, 1.5 percent. At one-half hour only one of the eight pupils showed an appreciable difference in contraction when a freshly prepared solution of carbachol was compared with a like nine months' old solution. The difference was one mm.

Two of the eight pupils at one hour showed a one-mm. or greater difference in contraction with the fresh solution when compared with the old solution. The greatest difference in contraction was 1.6 mm.

At two hours one pupil showed a difference greater than one mm. The amount was 1.2 mm. and the contraction was greater with the fresh solution.

The three-hour interval photographs revealed only one pupil of the eight which showed a contraction of one mm. or more. The new solution produced a contraction of 1.2 mm. more than the old solution.

At six hours two pupils showed a one-mm. or greater contraction with the freshly prepared carbachol than with the old.

At 24 hours two individuals showed a pupil that was still somewhat constricted, one having had the fresh solution and one the old.

There was no difference in the response to the miotic of the blue and brown irises.

A fresh solution was compared to a solution which had been frozen. After six hours a one-mm. greater contraction of the pupil was found with the fresh solution.

The fresh solution failed to produce at any of the six-hour intervals a greater contraction by one mm. than did a like solution which had been heated to 95°C. for one hour.

Reaction of the isolated albino rabbit iris sphincter

Freshly prepared solutions of carbachol chloride in 30 trials (table 3) produced contractions with a mean of 14.7 mm. ±6.31* and the 176- to 191-day (five to six months old) solutions in 22 trials produced contractions with a mean of 12.6 mm. ±7.60.* The actual difference between the two means was 2.1 mm. and the standard error of the difference between the two means 2.0. This resulted in a probability (P) of 0.2937. This means that, by sheer chance, differences as large as those reported will be observed 29 percent of the time.

The freshly prepared solutions of methacholine chloride in 35 trials (table 4) produced contractions with a mean of 18 mm. ±6.84* and the 192- to 223-day (six to nine months old) solutions in 21 trials produced contractions with a mean of 16.5 mm. ±8.00.* The actual difference between the two means was 2.5 mm. and the standard error of the difference between the two means 2.09. This resulted in a probability of 0.4777. This means that, by sheer chance, differences as large as those reported will be observed 47 percent of the time.

The action induced by a freshly prepared 1:2 million dilution of carbachol chloride was compared to that of a like solution which had been heated to 95°C. for one hour. The unheated solution produced contractions of 25, 28, 30, and 30 mm. and the heated solution contractions of 25, 29, 29, and 30 mm.

A 1:1 million freshly prepared dilution of methacholine chloride was heated to 95°C. for one hour and its action compared to that induced by a like unheated solution. The heated solution produced contractions

* Standard deviation.

TABLE 3

EFFECT OF CARBACHOL CHLORIDE ON THE ISOLATED RABBIT IRIS SPHINCTER

Dilution of Drug*	Millimeters of Contraction		Age in Days of Old Solution
	New Solution 30 Trials	Old Solution 22 Trials	
1:200 (T)	14	18	176
	12		
1:200 (M)	10	11	177
	10	3	
	9	4	
	11		
	13		
1:200 (M)	8	1	179
	13	1	
	11		
	9		
1:200 (T)	1	20	180
	20		
	21		
1:2 (M)	21	15	183
	17	15	
	16	16	
	16	7	
	16		
	14		
1:200 (T)	20	20	185
	20	20	
	28		
1:2 (M)	22	5	186
	18		
	22		
1:2 (M)	10	6	187
	0	0	
		17	
1:2 (M)	21	22	191
	20	5	
		21	
		20	
		21	

NOTE: Mean (new solution) 14.7±6.31 (standard deviation).
Mean (old solution) 12.6±7.60 (standard deviation).
* (M) indicates million. (T) indicates thousand.
Standard error of the difference between two means = 2.0 (102).
P = 0.2937 (85).

TABLE 4

EFFECT OF METHACHOLINE CHLORIDE ON THE ISOLATED RABBIT IRIS SPHINCTER

Dilution of Drug*	Millimeters of Contraction		Age in Days of Old Solution
	New Solution 35 Trials	Old Solution 21 Trials	
1:200 (T)	15	10	192
	12	21	
	21		
1:200 (T)	22	24	196
	24	13	
	24		
	26		
	26		
	4		
	7		
1:1 (M)	21	18	202
	22	17	
	7	16	
	9	16	
1:200 (T)	26	22	203
	24	26	
	25	27	
1:200 (T)	6	20	207
	16	6	
	18		
	17		
1:200 (T)	4	14	208
	14	21	
	22	24	
	22		
	21		
	7		
1:2 (M)	2	1	210
	21	1	
	12	23	
	22		
1:20 (M)	28	22	223
	18	4	
	21		
	19		

NOTE: Mean (new solution 18.0±6.84 (standard deviation).
Mean (old solution) 16.5±8.00 (standard deviation).
* (M) indicates million. (T) indicates thousand.
Standard error of the difference between two means = 2.09 (102).
P = 0.4777 (85).

of 14, 18, 21, and 22 mm. and the unheated solution contractions of 18, 19, 19, and 23 mm.

The response produced by a freshly prepared 1:2 million dilution of carbachol chloride was compared to that of a like solution which had been frozen. On one occasion the frozen and unfrozen solutions each induced a 14 mm. contraction, and on another the unfrozen a 29 mm. and the frozen a 30-mm. response.

Fresh solutions of methacholine chloride in dilutions from 1:200 thousand to 1:2 million were compared in action to that of like solutions which had been frozen. The unfrozen solutions produced contractions of

19, 21, and 23 mm. as compared to 22, 23, and 23 mm. for the frozen solutions.

Physostigmine sulfate freshly prepared in dilutions from 1:8 thousand to 1:1 billion failed to evoke a recordable contraction of the iris sphincter in 25 trials.

Freshly prepared neostigmine bromide in dilutions from 1:2 thousand to 1:200 million failed to cause iris sphincter contraction in 28 trials.

Pilocarpine hydrochloride in dilutions of 1:2 thousand to 1:200 thousand failed to cause an appreciable iris sphincter response.

Summary

The literature concerning drug solutions was reviewed with particular reference to the stability of miotics. Information was found to be meager as to how long solutions of miotics, as commonly prescribed, retain their initial pH, tolerate marked temperature change, and maintain adequate physiologic activity.

The pH of each solution was determined initially and every month up to 11 months thereafter. The pH change was minimal in spite of the fact that pilocarpine was the only one of the five solutions placed in phosphate buffer. Methacholine chloride showed the greatest change by becoming acid by 1.0 pH unit in 10 months. Physostigmine sulfate showed little fluctuation in hydrogen-ion concentration and remained color free for 11 months. The average pH of physostigmine, 3.32, caused the drug to be only mildly irritating.

Biologic assay was selected as a means of determining the physiologic activity of the solutions. This was accomplished by photographing the pupils of human subjects in whose eyes miotic drugs were instilled and by observing the action of miotic solutions on the isolated albino rabbit iris sphincter.

Less than half of the pupils photographed showed a greater response by one millimeter or more to freshly prepared 1.0-percent pilocarpine hydrochloride, 0.25-percent physostigmine sulfate, 5.0-percent neostigmine bromide, and 1.5-percent carbachol chloride solutions than they did to like solutions which had been stored for from eight to 10 months. Similar solutions which had been heated for one hour at 95°C. and those which had been frozen retained their ability to stimulate the iris as well as the fresh solutions. Methacholine chloride 20 percent in 1:3,000 benzalkonium chloride failed to constrict the pupils of normal human subjects.

Means are described of removing the rabbit's iris sphincter and directly recording its activity when bathed with various dilutions of miotics.

The irises of rabbits showed wide individual response to various drug dilutions. It was therefore necessary to find for each sphincter a dilution of miotic sufficient to produce a satisfactory maximal contraction.

It was observed that the albino rabbit's isolated iris sphincter responded best when suspended in a solution at 20°C.

Pilocarpine hydrochloride in dilutions of 1:2 thousand to 1:200 thousand, physostigmine sulfate in dilutions of 1:8 thousand to 1:1 billion, and neostigmine bromide in dilutions of 1:2 thousand to 1:200 million failed to cause recordable iris sphincter response.

Carbachol chloride freshly prepared in dilutions of 1:200 thousand to 1:200 million, in 30 trials compared with like five-to-six-months-old solutions in 22 trials, produced sphincter contractions with a mean of 14.7 mm. and 12.6 mm. respectively. Analysis of the figures involved in the experiment resulted in a probability of 0.2937 in 1.00 that such a difference will occur by chance. If a probability of 0.01 or less is used as indicating a significant difference in the contractions produced by the old and new solutions then the obtained P of 0.2937 reveals no significant difference in the physiologic activity induced by the new or five-to-six-months-old solutions of carbachol chloride.

Methacholine chloride freshly prepared in

dilutions of 1:200 thousand to 1:20 million, in 35 trials compared with like six to nine months' old solutions in 21 trials, produced sphincter contractions with a mean of 18 mm. and 16.5 mm., respectively. Analysis of the figures obtained in this experiment resulted in a probability of 0.4777. Such a probability indicates that there is no significant difference in the physiologic activity induced by the new or six to nine months' old solutions of methacholine chloride.

Conclusion

1. Aqueous solutions of miotics remain stable for as long as six months.

2. Pilocarpine hydrochloride, 1.0 percent in 1:5,000 benzalkonium chloride, sodium acid phosphate, and disodium phosphate showed no appreciable change in its pH of 6.36 and retained for eight months an ability to produce iris sphincter activity as determined by its effect on the normal pupil of human subjects. Pilocarpine solution was not affected in one hour by a temperature of 95°C. or by freezing.

3. Physostigmine sulfate, 0.25 percent in 1:5,000 benzalkonium chloride, boric acid, and sodium bisulfite retained a pH of 3.32 for at least 11 months and actively stimulated the pupils of normal human subjects after being stored 10 months. Physostigmine was not affected by freezing or by heating to 95°C. for one hour.

4. Neostigmine bromide, 5.0 percent in 1.0-percent boric acid, retained a pH of 5.84 for at least 11 months and actively stimulated the pupils of normal human subjects after being stored for nine months. Neostigmine was not affected in one hour by a temperature of 95°C. or by freezing.

5. Carbachol chloride, 1.5 percent in 1:3,000 benzalkonium chloride, retained a pH of 7.13 for at least eight months. The physiologic influence of carbachol, as determined by its effect on the pupils of normal human subjects and on the albino rabbit's isolated iris sphincter, was not appreciably diminished in six months nor materially changed by freezing or by a one hour temperature of 95°C.

6. Methacholine chloride, 20 percent in 1:3,000 benzalkonium chloride, had an initial pH of 4.55 and became more acid in 10 months by 1.0 pH unit. Solutions of methacholine which had been stored for as long as seven months and those which were heated to 95°C. for one hour or had been frozen were still as able as the fresh solutions to stimulate effectively the isolated iris sphincter of the albino rabbit.

1500 Medical Arts Building (2).

Complete references for this paper will be found on pages 397-400 of the March, 1954, issue.

Ophthalmic Miniature

A gentleman had a combat with a leopard which he had wounded. His right arm was most severely injured; he lost much blood, and great swelling followed. He had been for long before the subject of psoriasis. Within a fortnight of his injuries, whilst in bed and very ill, every trace of the psoriasis left him.

Jonathan Hutchinson,
Archives of Surgery, 1:384, 1890.

ORBITOCRANIAL PENETRATING INJURIES

EDWARD S. PLATT, M.D.
Auburn, New York

This report concerns a case of penetrating puncture wound through the right superior orbital plate into the brain with recovery and demonstrable weakness of the contralateral side. The patient was seen over a period of five and one-half years. A search of the available literature revealed the rarity of such an injury which made it seem worthwhile to review the previous reports and present the case.

REVIEW OF LITERATURE

Friemann[1] (1941) reported a case of perforation of the right globe and orbit by a splinter, probably iron, localized in the right temporal lobe by X ray. The eye was eviscerated "because of the danger of sympathetic ophthalmia." The patient was observed three and a half years after injury and was reported to have "no neurologic or psychologic symptoms except for a moderate forgetfulness and occasional conditions which might be construed as epileptiform mental absences."

Diachkov's[2] report on orbitocranial injury illustrates dramatically the contrast between present possibilities and those of the pre-antibiotic days. Diachkov presents eight cases and states that cranial involvement became apparent in two to four days after the injury as the result of abscess or diffuse meningitis. His first four patients died under conservative treatment. He ascribed recovery in the second four to exenteration of the orbit with conservation of periosteum, debridement and isolation of sinuses from the cranial cavity. He also had a fifth case seen two and one-half months after injury but no details were given. He felt that the only effective method of treatment was exteneration of the orbit.

In 1944, Slaughter and Alvis[3] reported a case of pneumo-encephalocele secondary to a puncture wound of the lid. A three-year-old child fell while carrying a pencil and received a wound of the right upper lid. He received local therapy and an unknown quantity of a sulfonamide for several days. On the 10th day he became drowsy and three days later was admitted to the hospital with a diagnosis of orbital cellulitis.

The eye was rotated down and out with slight proptosis. Localization occurred in the lid and Staphylococcus aureus was cultured. Six days after admission, a second abscess formed under the orbital rim and pus was found under pressure, as well as gas. Clostridium welchii was not found. After six more days the disc of the right eye showed indistinct margins with edema, dilatation and tortuosity of the vessels. The margin of the disc of the left eye was blurred but the changes were not so marked. There were no hemorrhages or exudates in either eye.

A cavity containing gas was demonstrated by X-ray films. This was checked twice a week and the gas had disappeared after one month in the hospital. The symptoms of brain abscess were minimal. The origin of the gas was not determined but was assumed to be caused by gas-forming organism implanted by the pencil. Recovery was complete.

In 1943, Lieux and St. Martin[4] reported a case of injury of the trigeminal nerve by a shell splinter which penetrated the orbit to the region of the gasserian ganglion and severed the ophthalmic nerve and the upper branch of the maxillary nerve. There was complete paralysis in the field of these two nerves but neuroparalytic keratitis was not seen and there was no change in the ocular tension or in the retinal arterial pressure.

An article by Kogan[5] stresses the value of co-operation between the ophthalmologist,

rhinologist, and neuro-surgeon, with which it is easy to agree. One wishes, however, that more information had been included in the abstract.

Rees[6] (1947) reported an injury to a three-year-old boy. A twig penetrated the left upper lid, orbit, and cranial cavity to the depth of the interpeduncular space. The globe was fixed, blind, and anesthetized due to injury to the ophthalmic branch of the fifth and to the second, third, fourth, and sixth cranial nerves. The twig was removed with forceps along the path of entry but the globe was destroyed in this procedure.

In 1949, McClure and Gardner[7] reported four cases of orbitocranial stab wounds with an excellent discussion of the anatomy and mechanism of such injuries. They also emphasized the ease of penetration of the orbital roof, a fact upon which the transorbital prefrontal leukotomy procedure of Freeman[10] is based. They contrast the mechanism of injury by high-speed projectiles, which tend to lacerate or rupture the globe, with injury by slower-moving objects, which may allow closure of the lid by a protective blinking reflex and also allow just enough more time so that the eye may be pushed aside into the orbital fat.

Their first case was that of a four-year-old boy who had fallen on a pitchfork tine which penetrated the inner part of the left upper lid. Drainage of cerebrospinal fluid through the wound was noted, vomiting occurred three times, and he complained of pain in the back of his neck. The lids of the left eye were swollen shut but the cranial-nerve functions were intact and reflexes were normal. X-ray interpretation was: "Localized changes involving superior orbital wall on the left—old trauma?" Penicillin was given, 30,000 units every three hours for five days, and one dose of tetanus and gas gangrene antitoxin was given. Leaking of cerebrospinal fluid stopped two days after admission and there were no sequelae.

McClure and Gardner's second case was that of a patient seen nine days after falling on stubble when he had immediately felt a flash of severe pain about the left eye and forehead. On arising he was unable to open the left eye and was told he had a scratch on the lower lid. There was complete paralysis of the left third, fourth, and sixth cranial nerves, and the ophthalmic division of the fifth. Vision was good, the fundus was normal, and X-ray films of the skull and orbit were normal. There was subsequent improvement but possible permanent partial external ophthalmoplegia, an unusual result from a minor wound.

Their third case was one of penetrating injury of the right upper lid, orbit, and into the brain by a large wooden splinter thrown from a rotary saw. The splinter was three feet long and was removed by fellow workmen at the shop. It was bloody for three inches, the probable depth of the penetration. The findings indicated a penetrating wound of the brain with internal and external ophthalmoplegia. There was no light perception and there was a complete flaccid left hemiplegia. Three months later the patient could move the eye in all directions but had no vision in it. He was able to walk but there had been only slight return of arm motion.

Case 4 of the series reported by McClure and Gardner was that of a patient who had been severely beaten and had received a stab wound of the neck on the left side. A diagnosis of stab wound of the neck and cerebral contusion with acute alcoholism was made. The right pupil was fixed and did not react to light.

The patient became rational by the third day but had developed marked swelling of the contents of the orbits. About the 12th day he lost all light perception in both eyes and had severe nasal hemorrhages. On the 14th day neurosurgical consultation revealed a small wound of the left lower lid which, with the clinical course, led to the additional diagnosis of a stab wound through the left

orbit into the base of the skull with laceration of the right internal carotid artery.

This diagnosis was confirmed by an arteriogram which disclosed a right carotid cavernous fistula. It was also proved at post mortem.

The authors emphasize the recommendation of Sweet and Bennett[11] that the carotid artery should be ligated above the cavernous sinus before ligating it in the neck, a procedure which was not possible with this patient because of his condition at the time of operation.

Mann[8] reports a case in which, during a fist fight, a pipe stem was driven through the orbit into the frontal lobe. Death followed in 48 hours from pneumococcic meningitis.

Schneider and Henderson[9] report a case of stab wound at the outer corner of the left brow, presumably by an ice pick. Later it was demonstrated that the weapon had penetrated at least 12 cm. from the place of entry. A confusing neurologic picture developed and the various signs were finally clarified at autopsy.

The immediate findings on examination in the emergency room were so slight that the patient was almost discharged without admission. During the first 24 hours, he gradually developed proptosis of the left eye with chemosis and a dilated fixed pupil.

Other signs of extensive damage developed gradually until death occurred on the eighth day. These signs included ipsilateral supranuclear facial palsy, hemihypalgesia and thermalgesia and contralateral involvement of the right 12th nerve, causing the tongue to deviate to the right.

The authors point out the value of neurologic evaluation of signs in contrast to the usual attempt to explain bizarre findings on the basis of diffuse subarachnoid bleeding.

Webster, Schneider, and Lofstrom[12] reported 40 cases of orbitocranial wounds, 20 of which involved one or both globes. When the defect of the orbital roof was extensive, they used fascial grafts for protection of the dural defect with later removal of the fascia, split-thickness graft, and reconstructive surgery. For the cases with small fragments through the orbital roof they advise an osteoplastic flap for adequate exposure and debridement. If brain abscess developed, they used the open treatment. When the globe was not disrupted they found vascular lesions of the eye in most cases with hemorrhages, retinal tears, and detachments.

King[13] reported two cases of encephalocele of the orbit, one following a right frontal craniotomy for orbital tumor, the other following compound fracture in the frontal region with comminution of the floor of the anterior fossa and protrusion of brain substance into the ethmoid sinuses and orbit.

While neither of these cases involved penetration through the orbit into the brain by a foreign body, the principles of repair and the prevention of subsequent encephalocele by the methods described would be most important in the event of extensive damage to the orbital roof by such an injury. King recommends fascia as a substitute for dura, and perforated tantalum for replacement of the floor of the anterior fossa. He credits the principles of this type of repair to a report by Dandy[17] published in 1929.

Two reports[14, 15] which were referred to by McClure and Gardner[7] have not been available to me for study and they are included in the references only to complete the list of cases which have been reported.

Ecker,[16] in a study of the bacterial contamination of penetrating wounds of the brain, demonstrated organisms which are commonly found in the soil and on the skin. His studies showed that "routine use of penicillin and sulfadiazine systemically maintained these bacteria in a state of contamination rather than that of infection for at least several days."

He recommended thorough debridement and water-tight closure in all possible cases

and stated that with such management "the most dangerous organisms proved to be the gram-negative bacilli which, however, were controlled when large doses of urea were administered as an adjunct to sulfonamide therapy."

Case report

History. On May 3, 1947, D. W., a three-year-old girl, received a penetrating injury of the right upper lid from the tine of a pitchfork when she stood up between the barn floor and the tail-gate of a box wagon just as her father swung a load of manure onto the wagon. She was last seen on November 10, 1952, giving five and one-half years for observation of the case.

Immediately after the accident, she was conscious but she "went to sleep" on the way to the office of the family physician, Dr. Albert Smith, who referred her to Auburn Memorial Hospital for observation. She vomited three times before admission and did not regain consciousness until three days later. There was no contributing history of preceding injury or illness.

On admission the child was pallid and comatose. There was a single puncture wound at the junction of the middle and outer third of the right upper lid with a hematoma of the upper lid closing the eye. There was a small amount of slightly blood-tinged fluid which drained slowly through the puncture wound for the first 48 hours. There was no evidence of direct injury to the globe and no visible penetration through the upper lid into the upper fornix. Both pupils were fixed and moderately dilated to six mm.

Normal reflexes were completely absent. Kernig's and Brudzinski's signs were negative but there was a positive Babinski on each side. There was generalized restlessness with frequent irritable crying. She vomited frequently for 11 hours after the injury.

A diagnosis of puncture wound, penetrating through the right supraorbital plate with presumably extensive brain damage, was made.

Supportive measures were instituted and antibiotic therapy was begun with 100,000 units of aqueous penicillin on admission followed by 50,000 units every three hours. Tetanus antitoxin, 1,500 units, was given after a negative skin test. Spinal puncture obtained bloody fluid at a pressure of 180 mm. of water. The day after admission Kernig's and Brudzinski's signs became positive.

On May 5th, the spinal tap was repeated and was found to be less bloody, with a red cell count of 63,750/cmm. and a normal ratio of white cells. On this day the tetanus antitoxin was repeated plus 2,000 units of gas-gangrene antitoxin.

At this time the skin test was positive and two injections of adrenalin 1:1,000, 0.5 cc. each, were given 45 minutes apart to control the skin reaction and the reaction at the site of injection. The left pupil responded well to light for the first time since the injury. In addition to the penicillin injections, sulfadiazine, 1.25 gm., was given intravenously followed by 0.5 gm. every four hours with 10 gr. of sodium bicarbonate.

On May 6th, she began to respond when her name was spoken and to take notice of her surroundings. She responded to pin pricks on the right side but not on the left. The dressing of the right eye was dry on the third day after injury.

On May 9th, both pupils reacted to light though that of the right eye was somewhat sluggish. There was no apparent ocular paresis. After this date, improvement was rapid and physiotherapy was begun for muscle re-education of the left arm and leg on May 12th, continuing until her discharge on May 21, 1947. Antibiotic medication was discontinued on May 12th and 13th.

Total dosage of penicillin in aqueous solution was 3,400,000 units. She received a total of 21.25 gm. of sulfadiazine.

The temperature ranged up to 102.6°F. on the third day with gradual reduction to

normal for the last two days. The pulse rate varied between 100 and 120 for the first six days after which it remained around 90. On May 20th, she began to walk with difficulty but without grossly visible residual signs.

Continued physiotherapy for muscle re-education of the left arm and leg was advised but was not followed up by the family.

In January, 1949, her physician, Dr. Smith, reported slight atrophy in the palm of the left hand and a tendency to dragging of the left leg with fatigue. Follow-up efforts were not successful until September, 1952, when she was brought to the office for preliminary ophthalmic examination as part of an evaluation of the residual effects of the injury.

At that time the left pupil was found to be one-mm. larger than the right pupil, both pupils reacting directly and consensually. There was a nearly invisible scar of the puncture wound in the right upper lid. The left pupil was somewhat sluggish in consensual reaction.

There was an exophoria of 10 prism diopters for near only, with a convergent near-point of 12 cm. and full rotation in all directions. Refraction revealed equal and normal vision with a low degree of hyperopia and astigmatism in each eye. No ocular paresis was noted.

Her mother said that she played actively with other children but that she seemed to stumble when running. She is reportedly slow in school work, especially reading and mathematics.

Muscle-function examination by Miss Ardis McCarty of the Physiotherapy Department of Auburn Memorial Hospital revealed reduction of muscle function to 50 or 75 percent of normal in all muscles of the left arm and leg. The previous recommendation for persistent muscle re-education was repeated and it is hoped that better cooperation will be obtained in attempting to prevent later lack of balance between the two sides.

Comment

A penetrating wound through the orbit and into the cranial cavity must be recognized as an immediate threat to life. Adequate doses of prophylactic serums and antibiotics should be started as soon as possible. Pediatric or medical care should be constant and complete with the addition of such other professional help as may be needed. This would include neurologic examination as recommended by Schneider and Henderson,[9] and McClure and Gardner,[7] and later physiotherapeutic measures when paralyses develop. It is noteworthy that signs of bilateral cerebral involvement may be observed because of an oblique angle of penetration.

The case herein reported is similar to the first case reported by McClure and Gardner,[7] although it presented evidence of deeper penetration as proven by the sequelae. Because of the critical condition of the child herein reported, no attempt was made to visualize the assumed injury by X-ray films of the orbit. The experience of McClure and Gardner[7] demonstrates the probability that no help would have been obtained from such a procedure in the presence of a known injury and definite neurologic signs.

Without direct observation of the degree of penetration of the pitchfork tine, it must be concluded that the point of the upward-curving tine reached the region of the right motor cortex or the motor pathway, giving residual weakness of the left arm and leg. It was assumed that a certain amount of meningitis must have resulted from the grossly contaminated tine and that this was adequately controlled by the combination of penicillin and sulfadiazine. The length of observation in this case appears to rule out the possibility of late abscess formation as suggested by Ecker[16] in his study of the bacteriologic factors.

In addition to the original admission as an ophthalmologic case, the care of this child required the combined efforts of pediatric, surgical, and physiotherapeutic services

of the hospital together with constant nursing care until the general condition began to improve.

No adequate explanation of the late variation in the left (contralateral) pupil can be offered. It is possible that this was a partial suppression of the sympathetic response of the right pupil to the stimulation attendant upon an unfamiliar procedure rather than a primary dilatation of the left pupil.

The good result in the case presented can be attributed to early recognition of the severity of the injury by the child's family physician, Dr. Albert Smith, and adequate pediatric care by Dr. Marshall Louis after admission to the hospital service, together with surgical consultation by Dr. G. P. Ross.

The ultimate outcome as affecting the mentality of the patient remains to be evaluated at a later date if co-operation can be obtained. The same is true of the muscular balance and co-ordination which could be better controlled by adequate physiotherapy and corrective exercises.

Summary

A case of penetrating injury through the right orbit into the cranial cavity with recovery and without injury to the globe is presented. There is definite residual evidence of damage to the motor region consisting of contralateral weakness of the extremities. The most likely area for this damage is the motor cortex of the right cerebral hemisphere above the level which would have produced facial paresis. It is remarkable that such deep penetration could be obtained without damage to the nerves transmitted through the supraorbital fissure and it seems probable that the pathway was through the orbital roof above the level of the cavernous sinus. A review of the recent literature is included.

National Bank Building.

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ROLE OF NICOTINIC ACID IN HEALING OF CORNEAL ULCERS*

LALIT P. AGARWAL, M.S., AND KRISHNA DATT, M.B.
Agra, India

Although corneal ulcers and their healing have received widespread attention since time immemorial and many and varied therapeutic agents have been used in an attempt to accelerate healing and lessen the density of the scar formed, not much work has been done so far on the role of nicotinic acid in healing these ulcers.

Nicotinic acid is an essential respiratory enzyme and, in its amide form, is an important link in various metabolic processes of the cell. The presence of nicotinic acid is therefore likely to increase cellular metabolism and hence regeneration. The other effect of nicotinic acid is vasodilatation which, again, can assist corneal wound healing by increasing metabolism by diffusion.

EARLIER INVESTIGATIONS

Casa[2] recommended the use of nicotinic acid and its compounds in the angiospasms of retinal arterial hypertension. Winso Rozzi[10] reported good results in toxic retrobulbar neuritis with nicotinic acid in combination with vitamin B_1.

Villa and Terchetti[4] said that the drug does not act by mere vasodilatation but also by increase in oxidation processes. The vasodilator effects have been reported by Loman[6] and Simonelle[8]; the latter also pointed out the vasodilator effects in ocular tissues. Simonelle[8] reports good results in various inflammatory conditions of the cornea and sclera.

Di Prima[7] from his animal experiments concludes that systemic administration of nicotinic acid accelerates the rate of healing and lessens the density of the scar following corneal ulcers. He ascribes the results to vasodilatation and increase in metabolic activity.

PRESENT INVESTIGATIONS

In 10 normal individuals, belonging to the class from which our hospital patients generally come, nicotinic-acid levels were estimated to serve as standard controls. These individuals did not show any clinical or subclinical evidence of nicotinic-acid deficiency. Their levels were comparable to the ones quoted by Bicknell[1] for normal individuals.

The present study was then divided into two main groups: (1) Experimental and (2) Clinical.

EXPERIMENTAL

The experimental work was undertaken to assess the role of nicotinic acid in the healing of corneal wounds. Albino rabbits, chosen as the experimental animals, were fed on an adequate nicotinic-acid diet. Two types of injuries were produced by the method adopted by Gundersen and Liebman[5]: (1) Superficial; (2) deep. There were three rabbits in each group, one of which, used as a control, was given no nicotinic acid. The other two rabbits were given 50 mg. nicotinic acid once daily until the corneal wounds healed.

CLINICAL

Out of the 57 cases of corneal ulcers studied, 20 cases were used as controls. In control cases, only routine treatment of corneal ulcers was given, that is, carbolization, penicillin drops (5,000 u./cc.), atropine ointment (one percent), and wet fomentations. In addition to routine treatment, 50 mg. of nicotinic acid daily was administered by the intramuscular route in 20 cases. Initial and final levels of nicotinic acid were estimated in each case. In the remaining 17 cases, 25 mg. of nicotinic acid were given subconjunctivally on alternate days in addition to the routine treatment.

* From the Department of Ophthalmology, Medical College of Agra.

TABLE 1

BLOOD NICOTINIC-ACID LEVELS IN NORMAL INDIVIDUALS*

Minimum	Maximum	Average
0.33 mg./100 cc.	0.79 mg./100 cc.	0.498 mg./100 cc.

* Estimated by the method of Swaminathan.[9]

OBSERVATIONS

In the experimental work and in Group III of the clinical cases, the healing time of corneal wounds in days and the type of scar formed were noted.

In Groups I and II of the clinical cases, the initial and final levels of nicotinic acid were noted in addition to the healing time and the type of scar formed. Tables 2 through 10 summarize the observations.

From the perusal of Tables 3, 4, and 5, it is evident that healing is accelerated in superficial corneal ulcer by either systemic or subconjunctival administration of nicotinic acid. The density of the scar is also less. Tables 6, 7, and 8 indicate that healing in deep corneal ulcers is also accelerated by the administration of nicotinic acid. The accel-

TABLE 2

EXPERIMENTAL

No.	Superficial		Deep	
	Healing (days)	Opacity	Healing (days)	Opacity
	Untreated with nicotinic acid			
1	5	Nil	7	Leukoma
	Treated with nicotinic acid			
2	3	Nil	4	Macula
3	3	Nil	5	Macula

TABLE 3

CLINICAL CONTROLS: CORNEAL ULCERS (SUPERFICIAL) NOT TREATED WITH NICOTINIC ACID

No.	Initial Level Nicotinic Acid (mg./100 cc.)	Final Level Nicotinic Acid (mg./100 cc.)	Healing (days)	Opacity
1	0.46	0.45	7	N
2	0.53	0.54	10	M
3	0.58	0.58	6	N
4	0.39	0.38	8	M
5	0.40	0.38	14	M
6	0.34	0.34	16	M
Av.	0.45	0.445	9.5	
Min.	0.34	0.34	7	
Max.	0.58	0.58	16	

TABLE 4

SUPERFICIAL CORNEAL ULCERS TREATED BY SYSTEMIC NICOTINIC ACID

No.	Initial Level Nicotinic Acid (mg./100 cc.)	Final Level Nicotinic Acid (mg./100 cc.)	Healing (days)	Opacity
1	0.43	0.45	10	N
2	0.56	0.56	10	N
3	0.49	0.52	5	N
4	0.41	0.41	14	M
5	0.72	0.73	4	N
6	0.67	0.67	4	N
7	0.37	0.39	5	N
8	0.41	0.41	3	N
9	0.38	0.39	6	N
10	0.32	0.36	12	M
11	0.38	0.38	10	M
12	0.31	0.33	11	M
Av.	0.455	0.466	7.75	
Min.	0.31	0.33	3	
Max.	0.72	0.73	14	

TABLE 5

SUPERFICIAL CORNEAL ULCERS TREATED BY SUBCONJUNCTIVAL NICOTINIC ACID

No.	Healing (days)	Opacity
1	7	N
2	6	M
3	2	—
4	6	M
5	6	N
Av.	5.4	
Min.	2	
Max.	7	

TABLE 6

CONTROL: DEEP CORNEAL ULCERS NOT TREATED BY NICOTINIC ACID

No.	Initial Level Nicotinic Acid (mg. /100 cc.)	Final Level Nicotinic Acid (mg. /100 cc.)	Healing (days)	Opacity
1	0.68	0.68	39	L
2	0.35	0.34	31	L
3	0.42	0.43	26	M
4	0.39	0.37	27	L
5	0.53	0.54	22	M
6	0.62	0.65	12	M
7	0.71	0.71	20	L
8	0.33	0.33	18	M
9	0.33	0.33	24	L
10	0.35	0.36	24	L
11	0.47	0.47	18	L
12	0.47	0.50	38	L
13	0.58	0.56	20	L
14	0.64	0.62	13	L
Av.	0.483	0.485	23.7	
Min.	0.33	0.33	12	
Max.	0.71	0.71	37	

TABLE 7

DEEP CORNEAL ULCERS TREATED BY SYSTEMIC NICOTINIC ACID

No.	Initial Level Nicotinic Acid (mg. /100 cc.)	Final Level Nicotinic Acid (mg. /100 cc.)	Healing (days)	Opacity
1	0.56	0.61	18	L
2	0.62	0.63	24	L
3	0.38	0.39	20	L
4	0.37	0.39	26	L
5	0.42	0.43	11	M
6	0.47	0.48	16	M
7	0.52	0.54	19	L
8	0.43	0.43	25	L
Av.	0.471	0.488	19.94	
Min.	0.37	0.39	11	
Max.	0.62	0.63	26	

TABLE 8

DEEP CORNEAL ULCERS TREATED BY SUBCONJUNCTIVAL NICOTINIC ACID

No.	Healing (days)	Opacity
1	5	N
2	12	L
3	14	L
4	9	L
5	11	M
6	9	L
7	8	L
8	8	M
9	13	L
10	10	M
11	9	L
12	12	M
Av.	10	
Min.	5	
Max.	14	

TABLE 9

NICOTINIC-ACID LEVELS IN BLOOD (mg./100 cc.)

Cases	Maximum	Minimum	Average
Normal	0.79	0.33	0.499
All ulcers	0.72	0.31	0.409
Superficial ulcers	0.72	0.31	0.453
Deep ulcers	0.71	0.33	0.48

TABLE 10

HEALING TIME IN DAYS

Cases	Average	Maximum	Minimum
Superficial ulcers			
Control	9.95	16	7
Systemic treated	7.73	14	3
Subconjunctival treated	5.4	7	2
Deep ulcers			
Control	23.7	39	12
Systemic treated	19.94	26	11
Subconjunctival treated	10	14	5

eration is more marked after subconjunctival administration.

DISCUSSION

Although there appears to be no deficiency of nicotinic acid in patients with corneal ulcer (table 9), since the averages in our study compare with those of Bicknell and Prescott,[1] our investigation would seem to indicate that nicotinic-acid therapy assists the healing process both indirectly and directly.

Indirectly, it appears to aid healing either by producing vasodilatation or by assisting cellular metabolism, or by a combination of

both factors. Under systemic nicotinic-acid treatment, healing time in the clinical cases herein reported was considerably reduced. This finding is supported by the work of Di Prima.[7]

One finding that seemed significant was that, in spite of rapid healing, congestion in treated cases persisted for a longer time than in nontreated cases, thereby pointing to the vasodilative effect of nicotinic acid. It would seem reasonable to assume, therefore, that nicotinic acid indirectly assists healing by: (1) Producing vasodilatation which (2) increases diffusion of metabolites, thereby (3) assisting corneal respiration and metabolism which (4) speeds regeneration of damaged corneal tissues.

Directly, nicotinic acid assists in tissue repair by virtue of its being a cohydrogenase I and II, coenzymes which are necessary to tissue respiration. These coenzymes indirectly assist the oxidation process by helping to withdraw hydrogen. The hydrogen so removed reacts with atmospheric oxygen to release energy. Di Prima[7] emphasizes this reaction and, in order to assess its role, we administered nicotinic acid subconjunctivally in concentrations sufficient to produce vasodilation. This further accelerated the healing process (table 10), indicating that nicotinic acid directly affects corneal healing in the way described.

In addition to accelerating healing—or because of it—our studies showed that nicotinic-acid therapy reduced scar formation. It has been stated by Duke-Elder[3] that, in deep ulcers, a thin layer of epithelium first fills the gap and then the corneal lamellae are formed. The explanation for thinner scars after nicotinic-acid therapy may be that epithelial regeneration has been accelerated to such an extent that the laying down of lamellae lags behind. However, this cannot, as yet, be stated with certainty.

Conclusions

1. Since nicotinic acid contains coenzymes essential to respiration, it directly aids cellular metabolism.

2. It accelerates corneal wound healing and reduces the healing time of corneal ulcers.

3. It also helps, in some unknown way, to reduce the density of scar formation after corneal ulcers. It may be that this is because epithelial regeneration is accelerated to a greater extent than the laying down of collagenous fibers.

4. By producing vasodilatation and increased diffusion, nicotinic acid increases corneal metabolism which, in turn, stimulates corneal healing.

Department of Ophthalmology,
The Medical College.

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LOCATION OF CONGENITAL DACRYOSTENOSIS IN CHILDREN

RICHARD WALDAPFEL,* M.D.
Grand Junction, Colorado

The embryology and developmental anatomy of the nasolacrimal system seems well established. It is generally accepted that the system develops in the naso-optic fissure from a thickening of the ectoderm which separates from the surface and buries itself as a solid rod of cells in the depth, forming the basis of the nasolacrimal duct; from the upper end of this rod the canaliculi are formed by outbudding and the sac by dilatation (Arey,[1] Broman,[2] Ask[3]).

Later, the solid rod of buried cells canalizes upward and downward and becomes patent. The last point to become patent is the lower nasal end—the opening of the nasolacrimal duct in the nasal cavity under the inferior turbinate. Patency at this point may often not be completed at birth. This results in persistence of a separating membrane between the inferior meatus and the nasal end of the nasolacrimal duct under the inferior turbinate in the newborn.

Regarded as the main cause of congenital dacryostenosis in infants, persistence of this membrane may cause epiphora, mucopurulent discharge, chronic dacryocystitis, and so forth (Schwarz,[4] Fiori-Ratti[5]). Cassady's[6] report, showing the anatomic-histologic conditions and the membrane separating the nasal end of the nasolacrimal duct from the inferior meatus in beautiful microscopic sections of noses of full-term stillborn infants, is quoted by Duke-Elder[7] who also gives an extensive bibliography.

Clinical findings herein reported do not, however, seem to corroborate this anatomic assumption. In order to locate the level of the dacryostenosis in children, lipiodol was injected into the stenosed tear passages. After X-ray films were taken, the usual probing and irrigation of the tear passages were carried out.

This routine preoperative procedure and the information obtained thereby are best described by reporting histories of nine cases.

CASE REPORTS

CASE 1

G. F., aged nine months, had had a discharge from the left eye since birth. At first the drainage was watery, then, when three months of age, it became yellow and thick and the lids of the left eye stuck together in the morning.

On examination, the conjunctival sac of the left eye appeared filled with pus. Pressure on the tear-sac region produced thick pus draining from the lower canaliculus. Medication of mild antiseptics and regular massage over the sac for one month did not change the condition.

Under slight ether anesthesia, after dilating the lower punctum sufficiently with the punctum dilator, a Bowman probe No. 2 was inserted and gently advanced in the direction of the duct until it reached the horizontal resistance of the nasal floor. Immediate gentle irrigation of the duct with saline solution yielded free drainage of the saline solution into the nose and throat.

Previous to the probing some lipiodol was injected into the lower canaliculus. It appeared very soon in the upper punctum. An X-ray film was then taken (fig. 1). It shows the lipiodol between the lids in the conjunctival sac and uniting in the internal canthus, then forming a small round shadow within the sac. The shadow ends below very near to the united canaliculi with a convex line. There is no lipiodol visible anywhere within the nasolacrimal duct or in the nose.

The child was dismissed home, was later seen twice, was fine and completely free from all previous symptoms of the dacryostenosis. No after care was necessary.

CASE 2

C. K., aged seven months, had the history that the left eye was watering and draining pus since birth. The right eye was normal.

The left conjunctival sac was filled with mucopurulent matter. Pressure on the left tear-sac area was followed by the same type of pus coming from the lower punctum. Massage of the left tear sac and the use of antiseptic eye drops for two weeks did not produce any change, nor did irrigation with saline solution.

Under ether anesthesia, lipiodol was injected into the lower canaliculus and soon appeared in the upper punctum. A Bowman probe was then introduced into the left lower punctum and advanced

* Deceased.

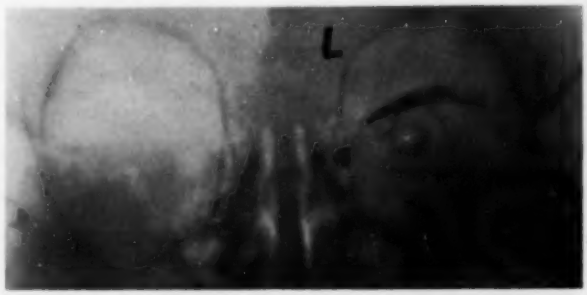

Fig. 1 (Waldapfel). Congenital dacryostenosis in left eye (Case 1). Lipiodol injected into the lower canaliculus. Its shadow is seen in the palpebral fissure and it ends within the tear sac with a convex line near the internal canthus. No lipiodol is visible in the nasolacrimal duct.

through the lower canaliculus into the sac, then down into the duct until the resistance of the horizontal nasal floor was felt.

Irrigation of the tear duct with saline could now be successfully performed. The injected liquid showed free passage to nose and pharynx.

The child was seen several times within the following week. Epiphora and draining of pus had completely disappeared and the child had no further trouble.

The preoperative X-ray film (fig. 2) shows the conjunctival sac filled with lipiodol which outlines upper fornix and palpebral fissure from the external to the internal canthus then fills the tear sac where it ends in the lower portion at the beginning of the tear duct in a cone-shaped line. No lipiodol appears in the lower sections of the duct.

CASE 3

R. D. P., aged three months. The mother reported that the child had "a cold in his left eye since he was four weeks old." The lids adhered most of the time and became red quite often.

The left conjunctival sac was filled with pus and the lids were covered with crusts. Massage of the tear-sac region on this side produced more pus in the conjunctival sac.

Again, before probing, lipiodol was injected into the lower canaliculus and appeared immediately in the upper punctum. After irrigation of left tear passages, the child was free from symptoms, and no further treatment was necessary.

The X-ray film taken before probing is shown in Figure 3. In spite of the double outlines caused by movement of the child, the lipiodol can be distinguished outlining the conjunctival sac, palpebral fissure, upper and lower fornix, and ending slightly beyond the inner canthus in the tear-sac region. No lipiodol appeared within the lower portion of the sac or in the nasolacrimal duct.

CASE 4

J. L. B., aged four months, had a watering left eye since birth. Some eyelashes were removed but that did not help. The lids of this eye were stuck together in the morning when drops and ointment were not used. The discharge from the eye was occasionally thick and yellow.

Examination revealed the right eye to be normal, the conjunctival sac of the left eye was filled with pus. Pressure on the left tear-sac area produced pus in both puncta.

Under light ether anesthesia, lipiodol was injected into the lower canaliculus and an X-ray film taken. Then irrigation and probing of the lacrimal passages of the left side were performed through the lower canaliculus in routine fashion. The fluid first came back through the upper canaliculus then drained permanently into nose and pharynx. The child was sent home and rechecked after one week and showed normal conditions with normal drainage of the tears.

The X-ray film taken before probing (fig. 4) revealed the conjunctival sac outlined in upper fornix and palpebral fissure by lipiodol which had collected in the internal canthus and then filled a very small portion of the sac. There is no visible trace of lipiodol within the nasolacrimal duct.

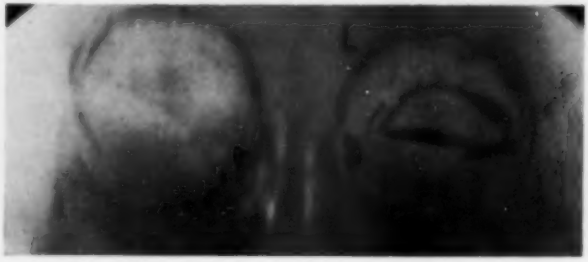

Fig. 2 (Waldapfel). Congenital dacryostenosis in left eye (Case 2). Lipiodol injected into the lower canaliculus outlines the upper fornix of the conjunctiva and the palpebral fissure, fills the tear sac, and ends with a cone-shaped line at the beginning of the tear duct. No lipiodol is seen in the lower nasal portions of the tear duct.

Fig. 3 (Waldapfel). Congenital dacryostenosis in left eye (Case 3). Lipiodol injected into the lower canaliculus outlines upper and lower conjunctival fornix and palpebral fissure and ends near the internal canthus in the tear sac. No lipiodol is seen within the lower portion of the sac or in the nasolacrimal duct.

Fig. 4 (Waldapfel). Congenital dacryostenosis in left eye (Case 4). Lipiodol injected into the lower canaliculus outlines the upper fornix of the conjunctival sac and the lid margin, the internal canthus, and is seen in small quantities within the tear sac. There is no lipiodol present within the nasolacrimal duct.

Fig. 5 (Waldapfel). Congenital dacryostenosis in right eye (Case 5). Lipiodol injected into the lower canaliculus appears in a small portion of the sac near the internal canthus, but not in the nasolacrimal duct.

Fig. 6 (Waldapfel). Congenital dacryostenosis in left eye (Case 6). Lipiodol injected into the lower canaliculus outlines upper fornix and lid margin, internal canthus, and a small area within the tear sac. No lipiodol is present within the nasolacrimal duct.

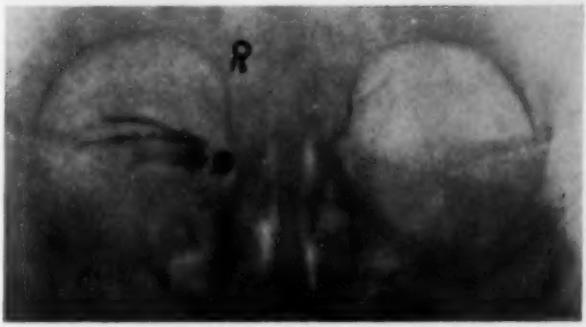

Fig. 7 (Waldapfel). Congenital dacryostenosis in right eye (Case 7). Lipiodol injected into the lower canaliculus outlines the lid margin and the internal canthus and fills a large portion of the tear sac. There is no lipiodol present within the nasolacrimal duct.

CASE 5

J. C. S., aged nine months, showed a right eye which, according to the mother's report, had been watering since birth with occasional drainage of pus. A conjunctivitis and blepharitis were present in the right eye, as well as flakes of pus mixed with the tears. Pressure on the tear-sac region produced a large quantity of pus.

The X-ray film taken after lipiodol injection into the lower canaliculus (fig. 5) is similar to Figure 4, the lipiodol appearing in the internal canthus and in a small portion of the sac. No lipiodol was present within the tear duct. Probing and irrigation were successfully performed and the child was dismissed without any further symptoms.

CASE 6

J. B., aged three months, showed a left eye which had been tearing since birth. Sometimes the eye became red and had pus in it. In the morning, the lids were stuck together and had to be cleaned. Another child had had the same condition and had to be operated on.

Examination revealed the left eye to be filled with tears. The pus in the conjunctival sac increased after pressure on the tear-sac region and each time drops of pus appeared at the puncta.

After lipiodol injection into the lower canaliculus, the X-ray film (fig. 6) showed the conjunctival sac of the left eye (L) to be outlined by the lipiodol which had collected in the internal canthus. It can also be seen in a very small area of the tear sac. No lipiodol was present within the tear duct.

Probing and irrigation cured the child permanently and no further treatment was necessary.

CASE 7

S. L. V., aged three months. The mother stated that the child's right eye had been mattering since birth and that recently the discharge had been pus. She was afraid the condition would eventually affect the eye.

Slight conjunctivitis and blepharitis were present in the right eye, with much pus in the lower fornix. Pressure on the right tear-sac region evacuated pus from both puncta.

Lipiodol injection through the right lower canaliculus and X-ray studies were done, then the right tear passages were irrigated and probed. The treatment proved to be an immediate success which remained permanent.

The X-ray film of this case (fig. 7) shows on the right side (R) the lipiodol outlining the lid margins and uniting in the internal canthus then filling a large portion of the sac, producing an oval-shaped contour. (A similar lighter shadow more temporally and below the internal canthus is caused by some lipiodol accumulation externally on the skin and

Fig. 8 (Waldapfel). Congenital dacryostenosis in both eyes (Case 8). Lipiodol injected in both inferior canaliculi outlines, on the left side, the upper conjunctival fornix and palpebral fissure and fills the tear sac from the level of the internal canthus downward. On the right side it fills the tear sac from the level of the internal canthus downward and, after a slight indentation, a small area in the upper portion of the nasolacrimal duct. There is no lipiodol visible in the lower sections of the duct on either side.

Fig. 9 (Waldapfel). Congenital dacryostenosis in right eye (Case 9). Lipiodol injected into the lower canaliculus is seen in the internal canthus and from its level downward within the tear sac. No lipiodol appears in the nasolacrimal duct.

extending slightly temporally along the infraorbital rim). There is no visible trace of lipiodol within the tear duct.

CASE 8

D. L. B., aged nine months had a history that both eyes had been inflamed most of the time since birth, the left eye worse. The general practitioner who takes care of the child had prescribed various drops and ointments for the child's recurrent conjunctivitis, with only temporary success and then referred the child to me.

Examination revealed a slight conjunctivitis in both eyes. A mucopurulent secretion was accumulated in both conjunctival fornices, more in the left. Pressure on both tear-sac areas produced pus in the puncta on both sides.

Lipiodol injection was done through the lower canaliculi in both eyes. The X-ray film taken immediately afterward (fig. 8) shows, in the left eye (L), the lipiodol outlining the upper fornix of the conjunctiva and lid margin, then filling the tear sac from the level of the internal canthus downward a short distance, producing a slightly cone-shaped shadow at the beginning of the tear duct, then ending. No trace of lipiodol is visible within the tear duct.

In the right eye (R), the lipiodol in the lower portion of the sac shows a slightly cone-shaped contour and then, after a slight indentation, its shadow continues in a straighter contour for a short distance downward, apparently also filling, on this side, a short portion of the tear duct. It terminates at a slightly lower level than on the left side. No trace of lipiodol can be seen in the lower sections of the tear duct.

Probing and irrigation of the tear passages in both eyes gave immediate success which was permanent. The mother reported in a checkup a week later that the right eye was still tearing somewhat the next day but slight massage of the tear-sac region on this side according to instructions eliminated even this faint residue of the previous condition. The child's eyes have been perfectly normal ever since.

CASE 9

F. J. D., aged seven and one-half months. The right eye had been watering since birth; lately, pus was draining most of the time. A conjunctivitis and blepharitis were present in the right eye. Slight pressure on the tear-sac area of this side produced pus which appeared in both puncta.

Figure 9 shows the lipiodol filling the internal canthus and a portion of the tear sac. No lipiodol is seen within the nasolacrimal duct.

Probing and irrigation of the right tear passages in routine manner eliminated all symptoms of the dacryostenosis. No further treatment was necessary.

COMMENT

In these nine cases of congenital dacryostenosis in children, aged three to nine months, all the well-known symptoms of this condition, epiphora, chronic dacryocystitis, conjunctivitis, and blepharitis, were present. In Case 8 the condition was bilateral. In Case 6 the condition was apparently familial as reported by Viallefont,[8] Peters,[9] and Lebouque.[10] In all cases, probing of the tear passages and irrigation of the involved side relieved the obstruction and permanently restored normal conditions.

According to the textbooks and to our present views, atresia of the nasolacrimal duct at its lower nasal end in the inferior meatus—due to a failure in canalization—produces a majority of cases of congenital dacryostenosis. X-ray examinations after lipiodol injection into the tear passages in a series of nine cases (10 obstructions) do not confirm that the stenosis is at the lower nasal end of the nasolacrimal duct. All these cases showed the stream of lipiodol blocked higher, at the upper end of the tear duct or within the sac itself.

In a search for an explanation for these findings and for the discrepancy between

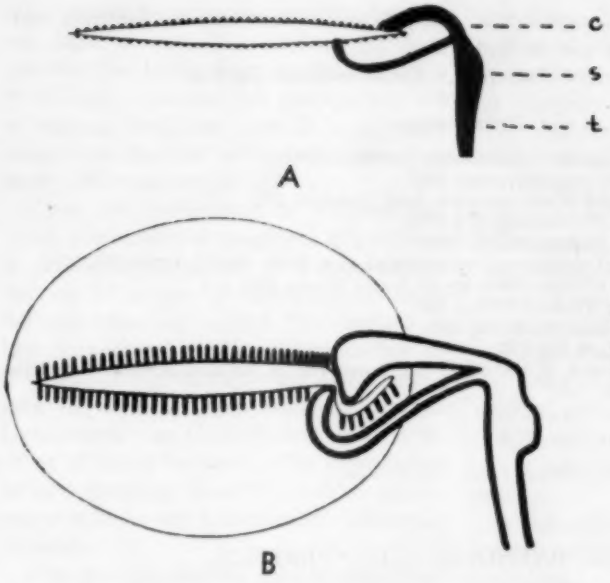

Fig. 10 (Waldapfel). Diagram of the development of the upper portion of the tear passages (modified after Ask).

(A) Solid epithelial rod indicating the anlage of the upper end of the tear duct (t), the tear sac (s) and the canaliculi (c). Canalization starts at s—t.

(B) After canalization.

them and the reported anatomic findings, the following interpretations may be considered:

1. Atresia may be present both at the lower end of the duct and at its upper end in the same case. Two cases reported by Becker[11] indicate the possibility of such double stenosis.

2. Atresia may be located at the lower end of the tear duct but the lipiodol does not penetrate farther down because of debris filling the nasolacrimal duct above the atresia.

3. The atresia develops solely at the upper end of the nasolacrimal duct and this, too, has its developmental foundation.

The solid epithelial rod, which separates from the ectoderm and is the first anlage of the tear duct, forms, by outbudding at its upper end, the canaliculi which are solid at first and canalize later (fig. 10).

Below the junction of the outbudding, canaliculi develop the tear sac by dilatation of the rod. It is in this region that canalization of the solid lacrimal anlage starts, proceeding downward and upward.

This area corresponds approximately to the site of the obstruction shown in our series of cases and a congenital stenosis at this place would indicate some failure in the canalization at its starting point.

If this theory can be substantiated, it would indicate another favorite site for lacrimal obstruction other than the nasal end of the duct in the inferior meatus. Apparently, obstruction of this site is frequent; in this series, it occurred in nine successive cases.

Further microscopic examinations with particular emphasis directed to the upper portion of the duct seem necessary; they may answer the questions raised by the findings reported here.

Summary

X-ray studies of the lipiodol-injected tear passages in nine cases of congenital lacrimal stenosis showed the stenosis, not at the lower nasal end of the nasolacrimal duct but in its upper portion. This does not support the generally accepted views which are based on findings furnished by developmental anatomic research.

Further study is needed to explain the clinical findings reported here and to find an interpretation for the apparent discrepancy between them and the anatomic findings.

De Merschman Gardens.

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CLINICAL PATHOLOGIC CONFERENCE

From the Laboratory of
PARKER HEATH, M.D.
Boston, Massachusetts

CASE 53-25

HISTORY

The patient, a 16-year-old girl, gave a history of a convergent left eye, sightless except for light perception until six weeks ago at which time she began to see. The known family history was not significant.

EXAMINATION

The left eye, normal in size, was convergent 25 degrees (Priestly Smith scale) and showed only slight limitation of movement outward; good movements in other fields. The anterior chamber was irregularly shallow, especially on the temporal side. A round pupil was displaced somewhat toward the 3-o'clock position; gave prompt reactions to light, but not equal on all sides of the arc. A cataractous lens was present and a rounded brown mass pushed the cataractous lens toward the temporal side. A small pupillary aperture could be seen.

Transillumination displayed a positive opaque shadow. Intraocular pressure (Schiøtz) was 25 mm. Hg. The pupil did not dilate readily with homatropine but appeared somewhat more distorted after the repeated use of homatropine. Through the small pupillary space the aphakic fundus could be seen. No gross changes were noted. The patient could readily count fingers at two feet and the use of a pinhole disc improved her vision. With a +10D. sph., vision was 20/200.

IMPRESSION

The temporal half of the iris was pushed forward by a mass behind. The rounded pupil was eccentrically displaced two mm. to the temporal side. The eye was suspected of harboring a tumor. Enucleation was advised and accepted.

*Discussion by Dr. Henry Allen**

The convergent squint and the symmetri-

cally ectopic pupil suggest that the left eye had basic abnormalities of long standing. On the other hand, vision of 20/200 with a +10D. sph. indicates that this eye was not entirely sightless from birth. It is not stated whether vision could be improved above this level with lenses or pinhole.

Upon this background of previous disorder, presumably of congenital origin, there is presented the picture of a mass behind the iris of an eye in which tension is at the upper limit of normal. The cataractous lens is apparently displaced laterally but no information is given regarding its size or degree of maturity. One wonders whether homatropine was the only drug used in an effort to dilate the pupil. The information given regarding transillumination is too vague to be of any assistance in differential diagnosis.

Four main possibilities must be considered in attempting to classify the mass behind the iris: congenital uveal cyst, hemorrhage beneath the uvea, intraocular neoplasm, and an inflammatory mass of the uveal tract.

There is nothing to suggest an inflammatory origin for the mass. The absence of adhesions, of keratic precipitates, and of vitreous opacities seems sufficient to exclude this possibility.

It is unlikely that a slow-growing tumor would have failed to produce glaucoma over a period of time. A tumor of recent origin would not be sufficient to explain the entire clinical picture. For these and other reasons, I believe that this eye does not contain a tumor.

The differentiation between a cyst of the ciliary body and a hemorrhage into or beneath the uveal tract cannot be made on the basis of clinical evidence presented in this case. My impression is that this eye will be found to contain a benign lesion with characteristics pointing to a congenital origin.

* Instructor in Ophthalmology, Harvard University; assistant at the Massachusetts Eye and Ear Infirmary.

Diagnoses

Clinical diagnosis. New growth of iris-ciliary body origin—possibly malignant in an amblyopic eye.

Dr. Allen's diagnosis. A benign lesion of congenital origin.

Anatomic diagnosis. 1. Intra-epithelial pigment-layer cyst of iris.

2. Atypical ciliary body processes with cyst formations attached to and in part composed of iris and retina, displacing lens toward the temporal side.

3. Early cataractous changes in the lens, the latter showing atypical structural proportions and posterior synechia with iris.

4. Persistent hyaloid artery.

5. Cystic degenerative changes in the retina.

6. Anterior-segment dysplasia with congenital malformation of the anterior uvea and retina.

Histopathologic description

Gross. Volume, 6.0 cm.; weight, 4.6 gm. Measurements: Vertical, 21.5 mm.; horizontal, 20.5 mm.; anteroposterior, 21.5 mm. Globe is small, rounded except for a 7.0 mm. bulge, rounded just anterior to the equator at the 10- to 12-o'clock positions.

Anterior segment: A 3.0 by 1.0 mm. superficial corneal abrasion at the 3-o'clock position, three mm. in from the limbus. Upper one half of cornea slightly cloudy. Pupil is round and regular (4.0 mm.) but eccentrically placed two mm. to nasal side. Temporal one half of iris is displaced forward. Anterior chamber is deep. A white-gray mass obscures the temporal one half of the pupillary space and is apparently lens which has been dislocated temporally with the nasal edge rotated posteriorly.

Posterior segment: Moderate amount of muscle bulk and blood clot attached. Optic nerve, two-mm. long. Eye opened horizontally. Cornea has usual thickness. Anterior chamber is deep.

Iris: Anterior surface is uniformly brown,

Fig. 1 (Heath). Whole eye, subiris tumor. (L) Displaced lens.

0.5 mm. in thickness. Temporal half is bulged forward with a thickened, yellow-opaque lens five mm. in anteroposterior diameter. Dislocated as described.

Lens: Ciliary-body angle is filled with deeply pigmented brown tissue. Hyperpigmentation of the ciliary body and a loss of light by transillumination from the 6-o'clock to 12-o'clock positions, extending back to the pars plana. The lens appears markedly adherent to the temporal iris leaf and ciliary body.

Vitreous: Mostly fluid with a few mucoid strands from the posterior retina to lens and ciliary body.

Retina: Attached throughout. The vessels from the disc are dilated. The macula is not remarkable. The choroid is slightly thickened posteriorly.

Microscopic. (Whole eye.)

Cornea. Slight differences in thickness of epithelium are noted. At the limbus is a degenerative pannus, moderate in extent. A few deep vessels are seen in substance of the stroma. The nuclei of the endothelium are pyknotic. There is an occasional red cell on the back of the cornea.

The *anterior chamber* is of average depth. The angle is open. The trabeculum contains pigment in granules and some cells. Also pigment is seen in the outer side of Schlemm's canal. Early anterior peripheral synechia are present in some meridians. Schlemm's canal is open, with rather a wide slit, and contains some red blood cells and protein-rich fluid. Pigment in the trabeculum is quite outstanding. It is also seen at the anterior border of iris, rather uniformly disposed. Pigment also extends into the head of the ciliary body and on the muscle bundles.

The *iris* stroma is loose, crypts are present. Pigment is scattered through the iris with the appearance of a brunette constitution. The sphincter shows an unusual amount of pigment on its anterior surface. Clump cells are noted in the iris. On the temporal posterior surface of the iris are noted some intra-epithelial cysts and, on the mesial border, an anomalous attachment of the retina to a cystic structure with a long peninsula of ciliary stroma extending behind the iris and as far over as the pupillary zone, in a position to push the lens over to the temporal side.

Running over the inner aspect of the ciliary body is a fenestrated membrane continuous with that from the pigment layer of retina and coupling with the choroid's Bruch's membrane—apparently a continuation of the latter.

As these structures run over the ciliary body, the choriocapillaris becomes occupied by larger vessels on the outer aspect of a

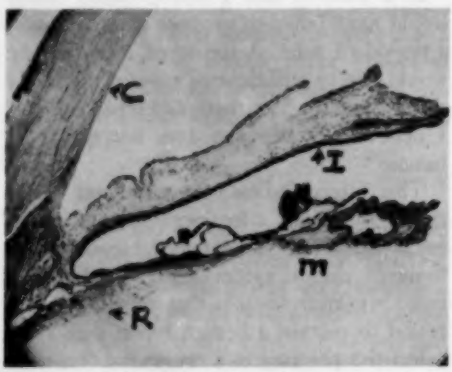

Fig. 2 (Heath). Detail of conglomerate tumor. (R) Retinal tissue. (M) Mixture of tissues, iris pigment epithelium above and ciliary processes below. (C) Cornea. (I) Iris.

glass layer; and on its inner aspect between it and a pigmented zone is seen the partial representation of an unpigmented retinal epithelium, normally part of the ciliary processes. This tissue continues inward toward the lens as a tongue or peninsula from the ciliary body.

The *retina* is represented in all these changes by typical retina up to the point of the ora. At the posterior aspect of the termination of the tongue of tissue from the ciliary body the structure becomes representative of the pigmented and nonpigmented epithelium of the ciliary processes with a heavy layer of pigment on its anterior surface and a pigmented knob of tissue lying in the region of the pupillary zone behind the iris.

The *vitreous* boundary behind this structure takes its usual course and position behind the lens. However, at the inner aspect of the lens is a remnant of the hyaloid artery which can be traced back in serial section to the inner side of the nervehead. The posterior pigment of the iris is continuous around this prolongation of tissue, somewhat diminished in thickness and containing intraepithelial cysts, reaching toward the lens and terminating in the knob, already mentioned, of pigment.

The *lens* is somewhat atypical in silhouette, the posterior surface is somewhat dished inward, and the mesial side is thinner than at the temporal border. On the anterior surface, the temporal region of the iris is attached to the capsule by posterior synechia.

The whole lens is displaced toward the temporal side. Some fluid clefts and the posterior extension of epithelial nuclei are well marked on the mesial side, but not so on the temporal side where the number of nuclei is diminished.

The *ciliary body* is rather thin and contains the pigment already mentioned. Larger vessels show some margination of cells. Alterations on ciliary body on mesial side have been described. The pars plana, as such, is very short, almost nonexistent, and the ciliary-

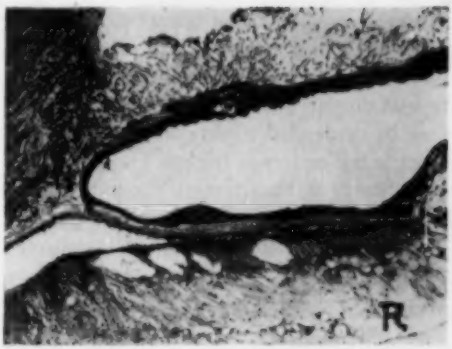

Fig. 3 (Heath). Detail of anterior displaced retinal tissue (R) attached to ciliary epithelial tissue.

muscle wedge enters very rapidly into the zone of choroid and retina.

On the temporal side, the ciliary body goes into the pars plana, and the ora of the retina is in its usual position backed by the choroid. The choroid is thin and attenuated due to fixation. From the mesial side it is thickened more than from the temporal and shows a relative increase in blood vessels.

The *choroid* has pushed up the retinal pigment layer in zones, creating small ovoid bodies and, in some of these pushed-up regions, occasiona. red blood cells are seen.

The *retina* has the usual number of ganglion cells in the macular region. These thin off in number rather rapidly toward the periphery. This is especially true on the mesial side.

The *nervehead* is reduced in diameter; the glial columns are irregular in thickness. A well-marked hyaloid artery remnant can be traced serially, breaking off just mesial to the nervehead and perhaps occupying the mesial rim of the nervehead at its real source.

Comment by Dr. Heath

The fundamental underlying situation in this case is related to a nonfamilial, unilateral, low visual acuity of congenital origin. The eye, normal in size, is convergent, displays a cataract, but retains active pupil reactions. The recent episode of improved

vision suggests displacement of the lens. Most of the miraculous, so-called, recoveries of vision in assumed-to-be blind eyes are due to lens displacement. In such a case the lens may be suspended weakly and a sudden jolt from a fall or minor trauma tilts, displaces, or subluxes it, thus freeing the pupil space.

In the case under consideration, another mechanism is demonstrable. A tumorous, opaque mass has forced the cataractous lens away from pupil space. The fact that vision was improved from light perception to 20/200 tells us that the cataract was advanced and opaque and that the lens is displaceable. We also learn that the fundus shows no gross lesion and that the best visual acuity obtainable is probably representative of an amblyopic eye.

Dr. Allen has considered this. For the etiology of the expanding opaque mass, he has indicated four possibilities. One of them is tumor. By tumor he means a solid, expanding neoplasm and this he rejects because of the absence of glaucoma and for the other reasons listed. However, malignant, solid tumor can appear behind or in the iris with or without increased intraocular pressure.

The very capable surgeon confronted with this case carefully considered nonmalignant and malignant possibilities. He was influenced toward a malignant classification by the rapid expansion of the solid-appearing mass, its obvious extent, and probable connections with the ciliary body as well as the iris. The shallowing depth of the anterior chamber also was a factor. The low visual acuity, the deforming nature of the growth before and probably after biopsy surgery were of significance.

The surgeon was aware of the probability that the presence of congenital defects would affect surgical manipulations adversely. Retinal separation would be likely. In short, he calculated that the risk from a radical attempt to save the globe was not justified considering the over-all low vision and probable amblyopia.

It is a likelihood that, in due time, continued expansion and growth of the congenital cyst would cause glaucoma in this case. Peripheral anterior synechias are already present also. The problems, after delay, would be the same as before, with the added complication of glaucoma to spur the surgeon to do something. A congenital lesion by process of nonmalignant growth or cystic expansion can destroy an eye just as inevitably as does the intraocular neoplasm.

243 Charles Street (4).

Ophthalmic Miniature

I have seen many persons whose brains have become heated in the spring by the smell of red roses.They get a catarrh and a running of the nose. They also had an irritation of the eye-lids, which when this season passed, subsided together with the catarrh and the nasal discharge. These people benefitted very little by treatment.

Baha'-Ul-Douleh, 1501
Quintessence, chapter 9.

NOTES, CASES, INSTRUMENTS

CONGENITAL EVERSION OF THE UPPER EYELIDS*

PAUL J. OSTRIKER, M.D., and
MORTIMER A. LASKY, M.D.
Brooklyn, New York

We are reporting a case of congenital eversion of the upper eyelids, a condition which we have been unable to find described in the ophthalmic literature. Also, unlike most other congenital lid defects that have been reported, there are no associated ocular or general anomalies.

There are a few scattered cases of congenital ectropion of the eyelids in the literature of the past 20 years,[1,2] but these are all associated with other anomalies and are quite unlike the complete eversion of the lids seen in this case. Ida Mann[3] mentions that congenital ectropion and entropion of the lids are exceedingly rare and makes no mention of congenital eversion.

Duke-Elder[1] states that "the lid margins may be everted secondarily as a congenital phenomenon in microphthalmos and in buphthalmos and in the case of the lower lid in the presence of orbito-palpebral cysts." He cites a case of Collins (1915) in which the lower lids were everted congenitally in a child with ptosis and epicanthus.

The following case, we believe, is interesting because of its unique nature, its subsequent normal course, and because of the theoretical question as to the mechanism involved in the lid eversion. The spontaneous subsidence and subsequent normal course appear not to implicate a developmental anomaly; the lack of any evidence of a specific infectious agent or abnormality of gestation or delivery seem to rule out these factors as well. At present we have no plausible explanation for the condition.

CASE REPORT

Baby S. was born on February 8, 1952, after a normal full-term spontaneous delivery. The mother, a 30-year-old gravida IV, para IV, had an entirely uneventful 36-week gestation. The Kline test was negative. Delivery was left occiput anterior with a 47-minute second stage of labor, and a 15-minute third stage. Pudendal block anesthesia with ether for crowning was employed.

The baby's color at birth was somewhat pale but there was no scalp or skull injury and no molding of the head. The birth weight was 8.5 lbs. Physical examination in the delivery room revealed a normal newborn except for thickened, swollen, injected, totally everted upper eyelids (fig. 1). Silver nitrate drops were not instilled into the eyes at birth or subsequently. Vital circumferences were: head 14.5 inches, abdomen 14 inches, and chest 14.5 inches.

Attempts to restore the lids to their normal position by manipulation were unsuccessful in the nursery on that same day. There was a minimal watery, clear discharge. Warm moist compresses were applied to the outside of the eyes. Aqueous penicillin, in doses of 100,000 u. was administered twice daily.

On the second postnatal day the condition

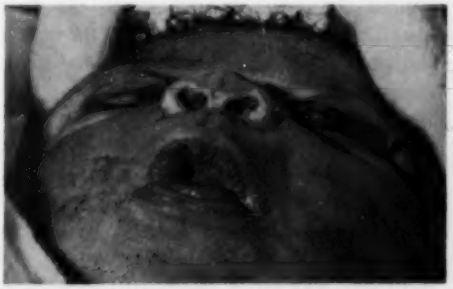

Fig. 1 (Ostriker and Lasky). Totally everted upper lids with marked chemosis of the conjunctivas and upper fornices and thickening of lid tissues.

* From the Department of Ophthalmology, Jewish Hospital of Brooklyn.

Fig. 2 (Ostriker and Lasky). Crying at the age of one year.

Fig. 3 (Ostriker and Lasky). Normal appearance at the age of one year.

of the lids remained unchanged. Scrapings from the conjunctival surfaces of the everted upper lids and secretion from the lids were studied for gonococcus, regular organisms, and virus inclusion bodies. Aureomycin ointment was instilled into the eyes and the lids were reverted and held in place with adhesive strips, the upper lids remaining quite swollen.

The bacteriologic reports of the smears were positive for pus cells and gram-negative bacilli, and E. coli was cultured from both eyes. No gonococcus appeared in smears or cultures and special staining techniques for inclusion bodies gave negative results.

Aureomycin ointment was applied daily and the lids were constantly held in place with adhesive strips. The swelling and injection gradually diminished and by the sixth postnatal day the upper lids, still slightly swollen, remained in their normal position without the adhesive strips for the first time. The baby was discharged on the eighth postnatal day with slightly swollen upper lids which were, however, in good apposition to the lower lids.

The baby was next seen in the out-patient department one month post-natal. Development was normal and the entire general examination revealed no abnormalities. Both upper palpebral conjunctivas were reddened and there was about one-fourth inch overriding of the upper lids over the lower lids on squeezing—that is, while crying. On examination, three weeks later, the lids were minimally swollen and injected and there was no overriding of the upper lids.

The baby was seen at the age of four months. There had been no spontaneous eversion of the upper lids since the baby was discharged from the hospital. There was slight swelling of both upper lids. The eyes closed tightly with the lids in good apposition. No evident defects in the tarsal plate were seen and the entire ocular examination including studies of extraocular motility, external eye and of the media and fundi produced normal findings.

The baby was seen again at the age of one year and the entire ocular and general examination revealed no abnormalities or significant findings (figs. 2 and 3).

Discussion

If this case represents a primary developmental anomaly of the upper lids (that is, a defect of the tarsal plate) it would be rather surprising that after a few weeks the lids should become entirely normal in appearance and function and remain that way thereafter. Yet there is no evidence of any causative infectious agent or of any unusual feature of gestation or delivery to account for the eversion.

An inclusion conjunctivitis, that could have been acquired during delivery, is unlikely as a causative agent in this case because of the five to 10-day incubation period

regularly seen with this infection, so that it almost never develops before the fifth postnatal day. In addition, no inclusion bodies were found in the lid scrapings, and inclusion conjunctivitis most frequently affects the lower lids.

E. coli found in smear and culture from the eyelids or cul-de-sacs of the newborn is not at all an unusual finding. It is almost never pathogenic. Friedlin and Loeb[4] report E. coli from the lid margins of newborn infants in 40 out of 100 cases in a routine survey. They believe the high incidence is probably due to contamination from the mother's perianal region during the delivery. These authors, as well as S. Whitmore, report even higher incidences for streptococcus and staphylococcus in cul-de-sacs of newborn infants, many of which organisms are potentially pathogenic. Yet none of these cases have congenital eversion of the lids.

Thus we have no satisfactory explanation for this condition.

Summary

A case of congenital eversion of the upper lids has been reported. There were no associated ocular or general anomalies. Within three weeks after birth the eyelids became entirely normal. A careful investigation for an etiology produced no positive results.

One Nevins Street (17).

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MELANOSARCOMA OF THE IRIS*

Report of a case

Vasilios M. Georgariou, M.D.
Athens, Greece

Of all the melanomas of the uveal tract that of the iris is the most rare. Komoto states a frequency ratio of 1:35,000. The first detailed account was given by Waren Tay (1886). The literature today contains known cases of sarcoma of the iris as reported by Fuchs, Morax, Wood, Pusey, Kurz, Zentmayer (1931), Mayer, Kubik (1933), Patterson (1935), Rosenbaum (1938), Vail (1946), Reese (1949), and finally by Joseph Laval (1952).

Dem. Polychronakos (Bull. Greek Ophth. Soc., 1950, p. 359) of the Eye Clinic of Athens University has reported a case of melanoma of the iris which has been confirmed histologically.

Etiologically some attribute special importance to eye wounds (G. Kosmetatos, Wah, Chan, Reese, Thon) and have related cases in which, after a period of 17 to 18 years since the eye had been wounded, there appeared a melanoma of the iris. Lane noted, in 66 out of 507 cases of sarcoma of the choroid, the appearance of a tumor following a wound.

Some accept the view that the melanomas of the iris already present can, after a lapse of years, develop into melanosarcomas (Hirschberg [1883], Wood, Pusey, Mayou). On the other hand Fuchs does not accept the degeneration of a melanoma to a malignant tumor.

Pigmented nevi of the iris also undergo malignant degeneration after a lapse of three

* From the Department of Ophthalmology, University of Athens Medical School.

to 25 years as reported by Nettleship, Albers, Wagener, Asbury and Vail.

Melanoma of the iris appears in the form of either melanosarcoma or leukosarcoma. The appearance of melanosarcoma is more frequent than that of leukosarcoma. Morax reported a frequency of appearance of 1:7. The melanosarcoma of the iris usually appears in persons between 10 to 75 years of age, irrespective of sex. It appears unilaterally, but a case of bilateral symmetrical melanosarcoma is reported in the bibliography (Kurz).

Melanoma of the iris first appears in the form of a pale yellow-brownish to dark black-brownish colored spot with clearly defined boundary; its size is that of a pinhead to five to six mm. and it first grows in thickness, not in area.

Sometimes it appears in the form of brown precipitates on the iris, or in the form of a nodule with protuberances which occupy the entire angle of the anterior chamber. At other times it is possible to distinguish pigmented granules away from the tumor which can be taken for daughter tumors in early development.

Most of them are rich in newly formed vessels which often form networks and when they burst frequently cause minor hemorrhages in the anterior chamber. These hemorrhages constitute one of the main clinical diagnostic signs (Mayou, 1932).

The melanoma is usually localized on the anterior surface of the iris and appears more often on the lower portion, less frequently on the upper, inner, and finally outer part of it. The localization varies, as it sometimes arises from the root of the iris, sometimes from Schlemm's canal, or the trabeculum, or even the stroma of the iris. Moreover, some authors have observed the appearance of a tumor of the iris on the border of a congenital coloboma. These tumors infiltrate all the layers of the iris first and metastasis occurs in the parts of the eye neighboring the iris.

The growth of the melanosarcoma of the iris is slower than that of melanoma of the choroid. As it extends it is capable of infiltrating the angle of the anterior chamber and the posterior surface of the cornea, but sometimes the infiltration is extended up to the substantia propria of the cornea, Schlemm's canal, the ciliary body, the choroid, and the retina.

Fehr reported a case of recurrence of sarcoma of the iris after iridectomy which infiltrated the cornea and the angle of the anterior chamber. More rare is the metastasis toward the sclera as well as that through Schlemm's canal, that is, the blood route of the outer surface of the globe (Gilbert).

The presence of a tumor on the anterior surface of the iris is, of course, easily diagnosed, but the differential diagnosis of other similar growths without inflammatory signs (angioma, posterior serous cysts, hyperplastic nevus, ectropion uveae of the pigmented epithelium, leiomyoma, encysted foreign body) is frequently quite difficult and the diagnosis is finally confirmed by histologic examination of the tumor.

The prognosis of melanoma of the iris is somewhat better than for other melanomas of the eye. It is the most benign, especially the spindle-cell type (Rosenbaum).

Dissemination of the melanoma via the blood stream is rare (Gilbert). Death occurring as a consequence of melanoma of the iris which was treated at an early stage has never been observed.

Case report

Patient K., a woman, aged 45 years, was admitted to the University Eye Clinic for consultation on October 30, 1952. Examination revealed a black-brownish, small tumor on the anterior surface of the iris of the left eye at about the 1:30- to the 3:30-o'clock positions, approximately the size of a lentil, bulging out of the root of the iris and not extending beyond the pupillary border.

According to the history this small growth was present since early childhood without undergoing any change. Twelve years previous to admission she had a temporary loss of vision due to the an-

terior chamber filling with blood which she thought was caused by the tumor. The patient was asymptomatic until one and one-half years ago when, at intervals of four to five months, a hyphema appeared in the left eye which reabsorbed in three to four days.

On examination by slitlamp, the iris tumor showed an anomalous berrylike surface, it was touching the posterior surface of the cornea, and in some areas it showed black, pigmented spots. It was very vascular, the vessels forming networks. A small hyphema was present in the anterior chamber.

Ophthalmoscopically the fundus was normal. Visual acuity was 20/20. The right eye was essentially negative.

The tumor was removed on November 4, 1952. A section of the cornea was performed on the limbus with a small keratome on either side of the tumor, joining together the two sections by means of scissors in such a manner that the tumor remained intact. A complete iridectomy was done which included the whole tumor, which did not reach the root of the iris.

Microscopic observation which was carried out during the operation did not show any dissemination of the tumor. The histologic examination revealed that it was a melanoma of the iris. It was composed of thickly packed collections of neoplastic cells ellipsoid, spindle or irregular in shape, most of which showed formation of melanin granules in their protoplasm. Such granules could also be found in the connective cells and between the mainly neoplastic elements. Only a few mitoses were observed. The blood vessels were numerous and in some places a radiating arrangement of the neoplastic cells about the canal of many of them was noted.

One month later, ophthalmoscopic as well as gonioscopic examination with digital pressure was again carried out and showed that for the present there was no spreading to the ciliary body or the choroid.

Discussion

In microscopic section the tumor is seen to be made up of spindle-shaped cells containing melanin and cells without it. In cases of leukosarcoma it is mainly composed of round cells. The vessels are abundant and frequently spongelike spaces filled with blood are observed. This is the reason why hemorrhages are so frequent. The pigment usually found is melanin existing either inside the neoplastic cells or inside the intermediate tissues. Apart from the melanin, hematogenous pigment, which is sometimes found in the leukocytes, is also observed.

Tumors which have been described in the literature (endotheliomas, peritheliomas) constitute a special form of melanoma. These tumors have been described mainly by Fleischer and Wintersteiner and are made up of cylindrical cells arranged in a rodlike manner, with a small amount of intermediate substance.

Conclusion

1. Melanosarcoma (malignant melanoma) of the iris developed on a pre-existent pigmented nevus. Malignant degeneration took place after about 25 years.

2. The relapsing hemorrhages of the anterior chamber constitute one of the main clinical diagnostic signs of the melanoma of the iris.

3. Whenever the case concerned is similar to the one described herein, without dissemination and glaucomatous symptoms and with normal vision, it is advisable to have the tumor excised, after a wide iridectomy in such a way that the tumor is untouched, a precaution which excludes the danger of dissemination.

4. During the operation, the section of the cornea must be carried out beyond the tumor and the section continued with scissors so that it is not touched. In this way the section of the iris can be carried out on each side up to the root of the iris so that the whole tumor is removed.

26 University Street.

TAKAYASU'S OR PULSELESS DISEASE

An unusual syndrome with ocular manifestations

William Charles Caccamise, M.D.
Rochester, New York
AND
Kunio Okuda, M.D.
Ube City, Japan

In 1908, Takayasu[1] reported a peculiar case with cataracta complicata and unusual ocular fundus findings. At the same meeting Onishi and Kagoshima reported two similar cases with the added observation of absent radial pulses in one of the patients.

It was not, however, until Shimizu's review articles,[2,3] that the disease became generally recognized by Japanese ophthalmologists as a rare but definite entity.

Symptoms

The classical symptoms are progressive peripapillary arteriovenous anastomoses, cataracta complicata, absence of palpable radial pulsations, and absence of detectable blood pressure in the arms. The majority of the cases have been in young women during and after puberty.

Other reported ocular findings have been obliteration of vessels in the periphery of the retina, mydriasis, iris atrophy, retinal and vitreous hemorrhages, retinitis proliferans, retinal detachment, dilatation of vessels of the bulbar conjunctiva and episclera, and very low central retinal artery blood pressure. Dodo[4] has applied the term "ophthalmo-angiopathia hypotonica" to these ocular manifestations.

In addition to the radial pulse and blood-pressure abnormalities the following non-ocular findings have been noted: Delicate skin, fine poorly pigmented hair, clubbed fingers (unusual), attacks of syncope attributed to hypersensitivity of the carotid sinus, intermittent claudication in the upper extremities, episodes of speech disturbance, attacks of pain in the orbit, eyeball, and chest, hemiplegia, hypo- and amenorrhea, perforated nasal septum (frequent), hearing defects, and increased sedimentation rate.

Etiology

These findings are allegedly the result of thrombosis of the subclavian and carotid arteries. Biopsy study by Shimizu revealed an arteritis primarily affecting the adventitia and media together with thrombosis. Histopathologically a tuberculous type of lesion was suggested but no organisms could be demonstrated.

Proposed theories for the etiology other than tuberculosis are Buerger's disease, allergy, panarteritis of unknown etiology, and collagen disease.[5]

Treatment

No effective treatment has been demonstrated to date. Attempts to reopen the carotid artery surgically have not been effective. Oishi[5] has theorized that cortisone and

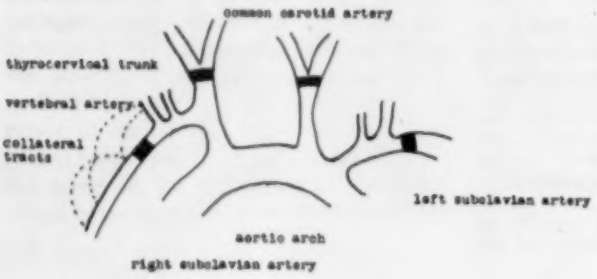

Fig. 1 (Caccamise and Okuda). Location of thromboses in a far-advanced case of Takayasu's or pulseless disease. A blood supply is maintained to the head by way of the vertebral arteries and the thyrocervical trunks. Collateral tracts to the arm were demonstrated by angiography.

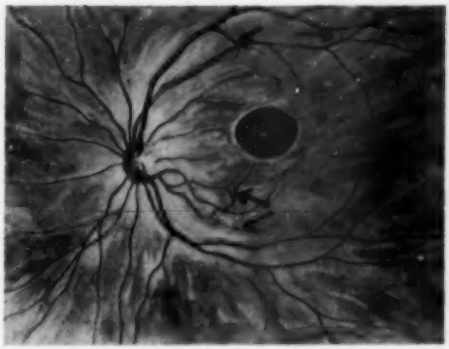

Fig. 2 (Caccamise and Okuda). Photograph of fundus drawing in an early case of Takayasu's disease. This is a view of the right eye as seen by indirect ophthalmoscopy. The arteriovenous shunts are the initial fundus manifestations of this bizarre syndrome. The left eye showed similar shunts. The patient is a 39-year-old Japanese in whom the diagnosis of Takayasu's or pulseless disease was made by S. Oishi, M.D., professor of ophthalmology at the Yamaguchi Medical College in Japan.)

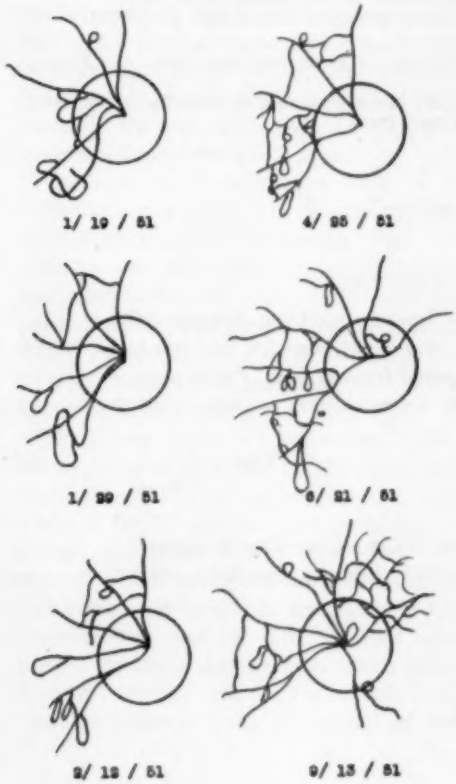

Fig. 3 (Caccamise and Okuda). Diagrammatic presentation of fundus findings in the right eye of Dr. Oishi's case of Takayasu's or pulseless disease. The term "anastomosis peripapillaris progressiva" has been applied to the progressive fundus changes. (Similar changes were evident in the left eye of this patient, a 39-year-old Japanese.) The view is that obtained by direct ophthalmoscopy.

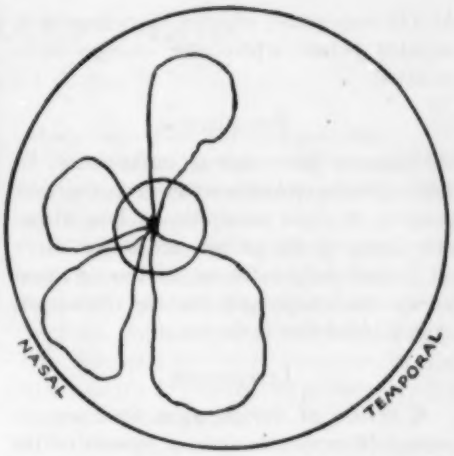

Fig. 4 (Caccamise and Okuda). Diagrammatic representation of fundus, O.S., in a 19-year-old Caucasian American girl. She presented the classical symptoms of this disease—loss of pulsations in the radial arteries, absence of detectable blood pressure in the arms, and ocular findings of peripapillary arteriovenous anastomoses and cataracta complicata. The conversion of the entire retinal vascular tree into simple peripapillary arteriovenous loop anastomoses is evidence of the far-advanced stage of the disease in this patient. There was no discernible difference in the appearance of the venous and arterial components of the loops. The disc was yellowish-white with fairly distinct edges. There was no apparent cupping. The papillary arteries and veins extended out from the disc onto the retina for a distance of one to two disc diameters. There each artery was seen to form a terminal loop anastomosis with the accompanying vein. This was visible inferiorly, nasally, and superiorly. There was no continuation of the retinal vessels beyond these loops. The choroidal vessels were only faintly visible peripherally through the retina. No hemorrhages or exudates were seen. The macula was clear without a foveolar reflex. A cataracta complicata obscured the fundus of the right eye. A similar cataract had previously been extracted from the left eye.

ACTH may prove of value if treatment is initiated before irreversible changes have occurred.

Prognosis

Prognosis for vision is unfavorable. In spite of successful cataract surgery, the cases progress to light perception or less. However, many of the patients are able to carry on limited daily activities, if the vertebral artery can compensate for the diminished carotid blood-flow to the brain.

Literature

A review of the Japanese literature revealed 58 reported cases. A review of the non-Japanese literature revealed only one case which was specifically reported as pulseless disease.[6] However, Aggeler and others[7] described a case which undoubtedly is an example of this condition. In their report mention is made of five additional cases from the literature that might be included in this group. A review of American ophthalmic literature failed to reveal any mention of Takayasu's or pulseless disease.

In an attemept to familiarize American ophthalmologists with the fundus findings in this disease, the accompanying sketches of the fundi in two typical cases of Takayasu's or pulseless disease are presented for the first time (figs. 1, 2, 3, and 4).

76 South Fitzhugh Street.
Yamaguchi Medical College

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Ophthalmic Miniature

Dear Sir, You know my laconic style. I never forget you. Are you well? I am so. How does Mrs. Cheselden? Had it not been for her, you had been here. Here are three cataracts ripened for you (Mr. Pierce assures me). Don't tell your wife that. Adieu. I don't intend to go to London. Good night; but answer me.

Yours,
A. Pope

Bath, November 21 (about 1740)
(Letter from Alexander Pope to William Cheselden, Surgeon.)
Recorded by John Nichols, *Literary Anecdotes,* 1812.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

CHICAGO OPHTHALMOLOGICAL SOCIETY

April 20, 1953

DR. WILLIAM F. HUGHES, JR., *President*

CLINICAL MEETING

(Presented by the Department of Ophthalmology, University of Illinois School of Medicine, DR. JOSEPH S. HAAS, *presiding.*)

COPPER WIRE IN LEFT EYE

DR. HAROLD Q. KIRK said that I. P., a 33-year-old white woman, entered the infirmary on October 6, 1952, complaining of clouding of vision and black "floaters" in the left eye, of two months' duration. She had been treated at another clinic for uveitis of unknown etiology, without improvement. Six months prior to this time she had been struck in the eye with a piece of wire. The eye cleared up within a few days.

The right eye was entirely normal.

Visual acuity in the left eye was 20/200, correctible to 20/70 with pinhole. The conjunctiva was moderately congested. Some fine dustlike keratic precipitates were deposited on the inferior half of the cornea. There was no evidence of perforation. The anterior chamber was deep with a moderately heavy beam and many cells.

The iris of the left eye had a more greenish appearance than that of the right, and was adhered to the lens by posterior synechias at the 11- and 6-o'clock positions, although the pupil dilated well. The lens showed a few peripheral cortical opacities and a nebulous opacity in the region of the anterior lens capsule. The vitreous was cloudy. A white, fingerlike mass with a broad base was seen to extend three or four mm. into the vitreous from the region of the ciliary body at the 4-o'clock position.

The fundus was seen with about 20/160 clarity and no other definite pathologic finding was visible. Tension was soft.

During the recent Academy meeting, the patient was seen by several members of the Board of Ophthalmology, and the following diagnoses were suggested: (1) Cysticercus, (2) diktyoma or other neoplasm of the ciliary body; (3) infectious granuloma; (4) intraocular foreign body.

A thorough uveitis survey was negative except for a three-plus, first strength purified protein derivative. Complement fixation tests for cysticercus and echinococcus were negative. Orbital films showed normal soft tissue density and no evidence of bony changes. Other tests were negative.

Atropine and cortisone therapy were given with no improvement. After three weeks' hospitalization, the patient was discharged but returned one week later. On re-admission, vision in the left eye was 20/100, not improved with pinhole. Further X-ray films were taken. A bone-free view on a dental film revealed a very fine rodlike, radiopaque foreign body in the region of the white vitreous mass. Attempts to remove this with the Berman localizer were not successful.

On November 13th, six months from the time of injury, a fine piece of copper wire was removed from the granuloma by forceps through a scleral incision. The wire was nonmagnetic and measured about two mm. in length, its diameter was that of a very fine needle. There appeared to be either a simple or combined detachment of the retina temporally. An elliptical scleral resection and prophylactic diathermy was performed after extraction of the foreign body.

Since surgery, the eye has gradually quieted down and the vitreous haze has partially cleared. The granuloma receded grad-

ually but vision decreased to 12/200. Retinal folds can be seen radiating from the region of the granuloma; however, a detachment, if present, remains localized.

The case is presented to emphasize the necessity of an accurate history and to demonstrate the value of bone-free X-ray studies for detection of small or nonradiopaque foreign bodies in the anterior portion of the globe.

Hydrops of the cornea

Dr. D. R. Magee presented R. L., a white man, aged 50 years, who came to the infirmary on February 16, 1953, complaining of "ballooning of the clear part of the right eye." There was a past history of epiphora and a similar condition of the left eye, which was enucleated in 1948 on the advice of two physicians.

The complaints referable to the right eye dated to early 1952, when he fell and sustained a minor injury to the eye and fractured a vertebra. Epiphora and burning of the eye were noted in April, 1952, and a contact lens was advised. The visual results were good until February, 1953, when the vision suddenly failed markedly. Epiphora and considerable photophobia were noted.

Examination showed vision in the right eye to be hand movements at three feet. There was moderate blepharospasm and photophobia with congestion of the globe and conjunctiva. There was marked ectasia of the cornea, inferior and nasal to the center, due to extreme edema of the stroma and epithelium, and bullous collections of fluid were seen between Descemet's membrane and the endothelium. Descemet's membrane showed many folds and rents.

Superficial vascularization extended from the limbus into the corneal stroma around the clock, most marked between the 2- and 7-o'clock positions where vessels extended to the central cornea. Peripherally the thickness of the stroma was greatly diminished. The anterior chamber was deep and clear.

The iris was blue and appeared normal. The pupil was three mm. in size, regular, round, and reacted normally to direct light. A faint red reflex was obtained. Tactile tension was normal.

The diagnosis of acute hydrops was made. Treatment consisted primarily of pressure dressings which were changed every other day, at which time White's A and D ointment and polysporin were instilled, as well as cortone ointment during the second and third week. Riboflavin (15 mg., three times daily before meals) was started the third week and has been maintained since.

He was discharged after eight weeks, at which time there was subjective and objective improvement. Minimal epiphora and photophobia were noted, and vision improved to finger counting at three feet. The corneal bulging and vascularization had decreased slightly. Epithelial and stromal edema were minimal and confined to the inferonasal periphery. The central cornea was slightly thicker than normal but the peripheral thinning remained unchanged. Moderate Descemet's folding also persisted.

Pulseless disease

Dr. Lawrence J. Lawson, Jr., presented a 45-year-old white woman who gave a history of having been well until six years ago, when she suddenly became weak and the right arm and leg became numb but not completely paretic. There was inability to express what she wanted to say which lasted 72 hour only, but the right hemianesthesia has persisted.

Her physician was unable to obtain her blood pressure or pulse and she was hospitalized for two weeks, during which time it was noted that the right optic disc was pale.

She was subsequently sent to a Chicago hospital with a tentative diagnosis of brain tumor and, after three weeks, was sent home with a diagnosis of rupture of a vein of the heart. She gradually recovered to the point

where she was able to do some housework.

Four years ago she struck her nose on the edge of a blunt object and became suddenly aware of only light perception in the right eye. Later that year she entered another Chicago hospital for treatment of vaginal bleeding. Physical examination and consultations revealed that the blood pressure and pulsations were not detectable in either arm, the left carotid or either dorsalis pedis arteries. Blood pressure in the lower extremities was 140/90.

Vision was recorded as: R.E., no light perception; L.E., 18/200. The right optic disc was waxy yellow with distinct edges and neovascularization in the region of the disc. There was no measurable elevation. General arteriolar constriction was present, with many small aneurysms or hemorrhages in the peripheral retina associated with peripheral choroidal atrophy. Segmented blood flow was noted in the veins. The impression was that of optic atrophy secondary to a vascular occlusion.

The left optic disc was slightly atrophic in appearance with minimal neovascularization. An inactive chorioretinitis was noted in the inferior temporal quadrant. The vessels were normal. Neurologic consultation concluded that the neurologic symptoms were compatible with a syndrome of the middle meningeal artery.

Because of a positive smear from curettement of the uterus, a diagnosis of adenocarcinoma of the uterus was made, and radium therapy was given. During her hospital stay extensive laboratory tests were made:

Encephalography showed cortical atrophy, enlarged subarachnoid spaces and basal cisterns, and enlarged ventricles; no shift. Skull and chest X rays were normal. The electrocardiogram showed sinus tachycardia. Fluoroscopy showed small traction diverticulum of the esophagus; moderate elongation and tortuosity of the aorta.

The electroencephalogram showed an abnormal record of moderate general slowing and generalized changes after hyperventilation. The direct arterial pressure (ulnar artery at the wrist) 45 cm. of water. Biopsy of the ulnar artery showed slight intimal sclerosis.

Blood studies were normal with the exception of the cholesterol which was 675 mg. percent; free cholesterol, 185 mg. percent and phospholipid, 17.5 mg. percent. It was found also that the blood cholesterol of the patient's sister, brother, daughter, and son were in excess of 280 mg. percent. Repeated cholesterols were: 446, 494, 540 mg. percent. Other tests were normal. The patient was discharged with a diagnosis of familial hypercholesteremia and adenocarcinoma of the uterus.

About 18 months ago, she developed an abscess of the right side of the nose which drained purulent material for a month and then subsided, but did not heal over, leaving an open fistula at the root of the nose.

Vision in the left eye gradually declined to light perception and on February 23, 1953, she suddenly became blind. When seen at the infirmary in March, there was no light perception in either eye. Intraocular pressure was 22 mm. Hg (Schiøtz). There was a two-plus flare in the anterior chamber and the iris was moderately atrophic with neovascularization in each eye.

The irises were adherent to the corneas peripherally for 360 degrees and an ectropion uveae was present. The pupils measured six mm. and were round and fixed. The lenses were completely opaque and obscured evaluation of the posterior segments.

Physical examination revealed that blood pressure and pulsations were not present in the upper extremities or right leg. There was a septal destruction of the nose. Exostoses were present over the knee and tibia, with firm, nontender nodules (cholesteatomas) over both Achilles tendons. Astereognosis was present on the right.

Other laboratory tests, including X-ray studies, were within normal limits with the

exception of the blood chemistry which showed cholesterol of 488 mg. percent and uric acid determination of 4.6. Her status is unchanged at the present time.

Caccamise and Whitman (Am. Heart J., 44:296, Oct., 1952) summarized 58 reported cases of "Pulseless disease" from the Japanese literature and reported one American case. Symptoms consist of (1) loss of pulsations of the radial arteries, (2) absence of detectable blood pressure in the upper extremities, and (3) ocular signs consisting of progressive formation of peripapillary arteriovenous anastomoses and cataracts.

The initial symptoms are syncopal episodes with or without convulsions, especially likely to occur when rising suddenly from the supine position or when turning the head, photopsia, blurred vision, or blindness.

The etiology is unknown. It has been postulated that it is a progressive thrombosis of the subclavian and carotid arteries secondary to a panarteritis of unknown etiology; or possibly due to a hypersensitive carotid body. The ocular findings of retinal arteriovenous anastomosis and cataract are thought to be the result of chronic vascular insufficiency.

Drusen of optic nerve

Dr. Carleton A. Keck presented R. C., a white man, aged 60 years, who was first seen on January 14, 1952, complaining of poor vision in the left eye and a feeling that he needed a change of glasses.

Vision at this time was: O.D., 20/25 with a +2.25D. sph. ⊂ −1.5D. cyl. ax. 38°; O.S., 20/50 with a +0.25D. sph. ⊂ −1.5D. cyl. ax. 105° (1948 prescription). He stated that there was a marked change in the prescription for the left eye in 1948.

Externally, the eyes were normal. The anterior chambers were clear. Pupils were normal in appearance and reflexes. There was moderate nuclear sclerosis in the right lens and marked nuclear sclerosis in the left lens with incipient cortical cataract changes in the lens periphery. The vitreous in both eyes contained some fine opacities but did not appear to be fluid in nature.

The fundus of the right eye was normal except for slight enlargement of the disc with irregularity and pigmentation of its margins. There was a normal macular reflex. The slight yellow cast to the disc was attributed to the nuclear sclerosis. Vessels were normal in appearance. No drusen were seen throughout the fundus.

The fundus of the left eye showed a greatly enlarged disc with marked irregularity and considerable pigmentary changes about the disc margins. The disc appeared to be slightly elevated in certain areas and had a marked amber or yellow color. The irregularities of the disc did not resemble opaque nerve fibers. The pigmentary changes resembled those seen following mild inflammatory reaction. The macula appeared to be normal.

Peripheral fields showed moderate contraction and an inferior nasal defect in the right eye. The left eye showed marked contraction and nearly total loss of the lower half of the field. A manifest refraction did not appreciably improve the vision. The left eye had become 1.50 diopters myopic due to nuclear sclerosis. Intraocular pressure was 20 mm. Hg in the right eye and 18 mm. Hg in the left eye (Schiøtz), and has been repeatedly normal since the initial examination.

Past medical history and laboratory examinations were nonrevealing.

Vision in the left eye on April 15, 1952, with the last correction (−1.5D. sph. ⊂ −1.0D. cyl. ax. 112°) was only 20/200, improved to 2/50 with a −3.5D. sph. ⊂ −1.0D. cyl. ax. 110°. The nuclear sclerosis is very marked and it is more difficult to view the left fundus clearly. The cataracts are slowly progressing and within a few years a cataract extraction may be necessary. The field changes in these cases are gen-

erally considered to be slowly progressive. (Lantern slides illustrated the field changes over a period of approximately a year.)

SCIENTIFIC PROGRAM

DR. R. TOWNLEY PATON, New York, presented a paper on "Modern trends in keratoplasty."

Richard C. Gamble,
Recording Secretary.

MADRID OPHTHALMOLOGICAL SOCIETY

March 13, 1953

RETINITIS PROLIFERANS AFTER TRAUMA

DR. ARJONA presented a patient who had suffered severe ocular trauma the preceding December, with complete loss of vision. The ophthalmoscopic picture was that of retinitis proliferans. The fundus showed two whitish "clouds" which surrounded the retinal vessels (above and temporally) and, in the periphery, resolved into an extensive reticular plexus. In the macular region could be seen a similar network which was slightly bulging (convex) as if it were cystic; in the center of which was a tiny hemorrhagic point.

MARFAN'S SYNDROME

DR. HERNANDEZ BENITO read a paper on Marfan's syndrome. He showed two brothers, one aged 13 years, the other seven years, both of whom showed bilateral luxation of the lens into the aqueous chamber, slight scoliosis, a funnel-shaped and narrow thorax, long, thin upper and lower limbs, arachnodactyly, little adipose tissue, and poor muscular development. The various pathogenic theories were reviewed—heredity, hormonal, congenital malformations, and lastly the theory of Weve.

Discussion. Dr. Arjona said that this syndrome and the syndrome of Marchesani were originally stated by the latter author to be entirely different, with different somatic manifestations. The opinion is not so rigidly held now as there are many overlapping symptoms. In both syndromes there is a weakness of the zonula associated with mesodermal dystrophy. This may lead to spherophakia or to ectopia lentis.

DR. H. BENITO closed the discussion by saying that one must heed the rule laid down by Maranon of the "multiplicity of congenital malformations." It is often very difficult to evaluate the symptoms as belonging to one syndrome or the other.

MECHANISM OF CHOROIDAL RUPTURE

Dr. Rio-Cabanas, after discussing the various theories of the production of traumatic ruptures of the choroid, said that no one mechanism can explain all cases. The various mechanisms should all be explored. These include the optic nerve which like a semirigid stem opposes the displacement of the eyeball, possible rupture of the ciliary vessels, the role of the choroidal vessels, sudden traction by the recti and oblique muscles, and the curbing action to displacement of the globe by the opposing pull of the vessels and nerves which enter or emerge at the posterior pole, as well as the opposition by the walls and contents of the orbit, all of which together may explain rupture of the choroid.

Discussion. DR. ARJONA said that he wanted to add one more factor to those mentioned, one of great value and propounded by Saemisch. It is the influence which the posterior ciliary arteries and the vorticose veins have in holding the choroid and preventing its displacement when the counteracting forces are working. They act somewhat like vault-supporting arches between which are weak zones which break (and always present an arched form) because of the equatorial traction which the flattening of the eye produces.

Dr. Mario Esteban said that there were references to chorioretinal lesions produced at a distance by explosive waves, the results of the air vibration and shock without any traumatism by contact with the globe. In his *Atlas of Ophthalmoscopy of the War*, Lagrange has described many such lesions, the most frequent being laceration of the choroid at the posterior pole.

Dr. Marin Amat said that the pathogenesis of rupture of the choroid involved many factors, the most numerous of which were contributing factors; others act as constant elements. Of these latter, there are two:

1. The counterstroke which is observed in contusions of the globe of moderate intensity and in contusions where only the retina seems to be affected (Berlin's edema). The edematous zone is greater in the region of actual trauma than at the opposite end (counterstroke action), except when the injuring missile strikes anteroposteriorly. Then the zone of greatest edema is in the zone of the counterstroke. If the contusion is very violent, the effects of the counterstroke will involve the choroid.

2. The second is a logical sequence of the first. As the eyeball is flattened and the sclera yields, the retina (except the part of the retina attached at the papilla and at the ora serrata) and the choroid have to slide on it. Since the choroid has its principal attachments to the sclera in the region of the penetrating posterior ciliary arteries, in the region of the posterior pole of the eye, and at the vorticose veins near the equator, but to a greater extent at the place of penetration of those arteries, it is easily seen that, as the choroid cannot yield, it will break in this region.

It is probable that the short posterior ciliary arteries are more numerous in the temporal zone because they have to supply the macular region and the retinal circulation is poor there. This may explain why the rupture is most common between the macula and the papilla.

Dr. Rio Cabanas said that he was more in agreement with the theory of Hudelo. But this too cannot explain all cases of arcuate rupture of the choroid. He had seen a case in which the clinical picture accorded with the theory of Hudelo, but it is necessary to postulate accessory factors, some of which predominate in one case, others in another case. The role of the optic nerve may not be dominant, but he believes it plays some part. Unquestionably one of the fundamental factors in the vascular network which fixes the choroid and prevents its displacement explains many of the cases of rupture of the choroid. But he emphasized that all cases of traumatic rupture of the choroid cannot be ascribed to one theory or to one cause. Many jointly acting factors are involved.

Joseph I. Pascal,
Translator.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

February 19, 1953

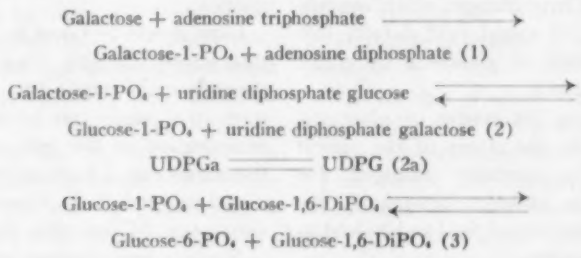
Dr. George F. J. Kelly, *Chairman*

Galactose cataracts

Dr. Edward Cannon and Dr. Joseph Ritter (by invitation) discussed the case of a four and one-half year-old girl with galactose cataracts. Her main signs and symptoms, in addition to zonular cataracts, were hepatomegaly, retarded physical development, elevated total blood sugar, and melituria. The specific sugar in the urine was identified by paper chromatography. Discission was performed on one cataract, and the other did not regress after the child was on a milk-free diet for eight months; other symptoms did regress.

Galactosemia is considered an inborn error in the metabolism of ingested galactose. Galactose diffuses readily through body tissues and the physiopathologic aberration of various body structures and functions which is noted in this entity is probably on a toxic basis. There are considerable experimental data in the literature concerning in vivo and in vitro effects of galactose on rat lens.

Discussion. Harry Green, Ph.D.: Our knowledge of the metabolic behavior of galactose may be summarized by the following equations:



The enzymes involved in the above reactions are, respectively, hexokinase, phosphogalactoisomerase, and phosphoglucomutase, while adenosine triphosphate, uridine diphosphate glucose, and glucose-1,6-diphosphate are the corresponding coenzymes. The product glucose-6-phosphate is subsequently metabolized in accordance with the classic glycolytic scheme of Embden and Meyerhof to pyruvic and lactic acid. Whether similar reactions occur in the lens is not known.

In the normal lens the major source of energy is glucose, although the metabolic pathway that glucose travels to lactic acid in the lens is still not completely known. Muller, in 1937, found that with bovine eyes, as the age of the lens increases the rate of glucose disappearance is diminished. With the inception of cataract formation this rate is further decreased until in a mature cataract hardly any carbohydrate metabolism is measurable. It is reasonable to assume, therefore, in our present state of knowledge of lens metabolism, that a slowing down of the normal rate of glucose utilization by the lens for whatever reason may predispose to the development of cataract.

The reactions just described were examined for the purpose of speculating as to the ways in which excess galactose or the accumulation of galactose-1-phosphate could disrupt the sequence of events.

It was tentatively suggested that in the normal lens small amounts of galactose are readily metabolized, presumably in accordance with these reactions. In galactosemia, however, the normal carbohydrate metabolism may be interfered with at any of the indicated stages, thereby causing a derangement in the mechanism for the generation of energy necessary for protein synthesis, fat metabolism, growth, maintenance of transparency, and so forth.

Simulated progression of field defects in glaucoma

Dr. Robert M. Day (by invitation) presented an experimental and clinical study of three factors which may cause simulated progression of the visual field defects in glaucoma. These three factors are:

1. Loss of transparency of the ocular media (cataract formation, particularly of the diffuse nuclear type).

2. Artificial myopia associated with ciliary spasm which results from the use of miotics in nonpresbyopic eyes.

3. Miosis. All of these factors cause a reduction in the intensity of the retinal stimulus by the test object.

The following conclusions were reached:

1. Visual fields of nonpresbyopic patients with glaucoma should not be taken until after any accommodative spasm induced by miotic therapy has subsided.

2. In charting the visual fields in glaucomatous patients, the pupils should, ideally, be of the same size during subsequent examinations.

3. Increasing myopia associated with decrease in the size of the visual field in glaucomatous eyes suggests the progression of lens changes.

4. Increasing lens changes often explain the progression of visual field defects following the control of glaucoma by either miotics or surgery.

5. In following the course of glaucoma with visual fields, the status of the optical media should be carefully evaluated, for opacities (that is, cataract formation) may cause an apparent loss of field and so lead to unwarranted surgery.

Discussion. Dr. Harold G. Scheie: The greatest benefit to be derived so far from the arguments between advocates of the neurovascular and the gonioscopic schools of thought regarding glaucoma has been in the management of the disease.

Both schools emphasize the importance of distinguishing between acute and chronic glaucoma, and there is increasing agreement that a different therapeutic approach should be employed for each type. Surgical intervention is advised in acute congestive glaucoma, while chronic simple glaucoma should be treated medically as long as damage to the eye can be prevented by this means.

The prime indications for surgery are uncontrolled tension or increasing visual field defects. Since the ocular tension and visual fields are of such importance, misleading results from visual field studies are as unpardonable as inaccurate tonometry.

We should be thoroughly familiar with any factor which might simulate progressive visual field loss and serve as a false indication for surgery. Dr. Day has mentioned refractive errors, miosis, and cataractous changes, all of which can cause apparent increase of visual field defects by reducing the intensity of the retinal stimulation from a test object of given size. He has mentioned the precautions which should be taken, such as recording the visual acuity in younger people in whom artificial myopia might be induced by miotics. He also mentioned the wisdom of recording the size of the pupil and urged taking visual fields at the same interval of time after instilling miotics.

Care should be taken to recognize nuclear cataractous changes. This may be difficult through small pupils but, if the angles are open, the pupils can be safely dilated for examination of the lens and evaluation of the nervehead. I have never seen increasing visual field loss in the absence of progressive excavation of the optic nerve.

Nuclear lens changes are usually accompanied by increasing myopia. One's judgment should balance these against other clinical signs such as the level of tension, age of the patient, and other factors. Emphasis is to be placed on avoiding surgery in this type of glaucoma whenever possible.

Increasing lens changes very frequently cause apparent loss of visual fields following normalization of tension by filtration procedures. We have seen several patients in one glaucoma clinic whose eyes seemed to have continued to deteriorate following normalization of tension by corneoscleral trephination. However, they all showed increasing myopia due to cataractous changes, as shown by Dr. Day, and following cataract extraction the visual fields returned to their original state. This serves as an argument against progressive loss of visual fields of a glaucomatous nature from purely vascular degeneration.

Dr. George F. J. Kelly: Dr. Day's presentation has been most interesting and very constructive. He has emphasized the im-

portance of the proper evaluation of visual fields.

As he has stated, many times in glaucoma surgery it has been felt that the visual field lessened even though the tension was reduced and stayed reduced, whereas the true situation was that the visual field had remained the same but was not properly surveyed.

I think the findings in an eye that had cataractous changes in 1939, with an arcuate scotoma and then, in 1949, following operation, no arcuate defect, are most unusual. The only explanation for this, as I see it, would be that the arcuate defect in 1939 was relative. If that defect was absolute, and most arcuate defects are absolute, there should be no recovery.

With regard to the contraction of the internal isopters, causing baring of the blindspot, following the use of pilocarpine, I wonder whether the given explanation, namely miosis, explains the whole story. One would also have to think that maybe pilocarpine has some action on the vasomotor system.

I have seen many times in mild cases of glaucoma, where the visual fields showed a baring of the blindspot, that, following the use of pilocarpine, the internal isopters would be larger and no baring evident.

Dr. Robert M. Day: I would like to thank the discussors. In answer to Dr. Kelly, my explanation for the disappearance of the arcuate scotoma when the cataract was removed was that the same size objects were used. A cataract in effect decreases the stimulation of the retina. In other words, it has the same effect as using a smaller test object. The arcuate scotoma was not charted, but my interpretation would be that it is still there and could be found with a smaller test object. As to the improvement of the field which occasionally occurs following the control of tension, I am sure that another factor is responsible for that. This factor was not taken up at all in this discussion.

Cataracts in alloxan diabetes

Dr. David Naidoff, Dr. Irwin J. Pincus, Dr. Arno E. Town, and M. E. Scott, B.S. (by invitation): Alloxan was injected in varying doses and varying rates intravenously into 107 rabbits. Of the 67 animals which survived after five days, 32 developed definite diabetes.

Lens changes were observed in every diabetic animal and these changes were seen in both eyes in which they were of equal severity. It is felt that the lens changes occur in the first 24 to 48 hours after the injection of the alloxan and, when first seen, appear as an increased density or faint striations in the equator.

The changes progress in a definite pattern and, at the end of the first week, can be seen as tiny vacuoles in the equatorial zone in the cortex. The vacuoles in turn appear to coalesce, more opacities form, and eventually, by the 16th week, the lens is so cloudy, in severely diabetic animals, that the fundus cannot be seen. Slitlamp examination shows the changes to be most marked in the posterior cortex, and about the equator.

Frequent examinations of the rabbits showed that the lens changes varied remarkably from day to day. Although the pattern of the cataract development was constantly changing, a definite progression did occur, and its extent and severity apparently depended upon the severity and the duration of the diabetes.

These diabetic animals showed lens changes, the degree of which fluctuated very greatly, and concomitant observations of the blood-sugar levels and urinary-glucose outputs revealed striking fluctuations at the same time. These lens changes were of such a degree as to give the impression that, up to a certain point, they may be reversible.

Discussion. Dr. Irving H. Leopold: As you know diabetes mellitus is not a disease entity but a syndrome characterized by hyperglycemia and glycosuria. So defined, diabetes mellitus can be produced by several

experimental methods. This picture can be produced by removing the pancreas in experimental animals.

Recently, when the pancreas of humans who suffered malignancy of the pancreas were removed, the clinical symptoms of diabetes mellitus resulted. The findings closely resembled those found in experimental pancreatectomy and in clinical diabetes, but the insulin requirements rarely exceeded 25 to 40 units per day for a patient maintained on a diet of 250 gm. of carbohydrate, 100 gm. of protein, and 100 gm. of fat.

This may signify the existence of a hyperglycemic factor in the pancreas, which is necessary to bring about the high blood-sugar levels that occur in clinical diabetes mellitus. Such a factor has been isolated from impure insulin and from the islets.

By injecting anterior pituitary extracts daily and increasing the dose every few days, one can produce, in a dog, a diabetic state which persists indefinitely, although the injections are discontinued. The treatment must be continued for a few weeks, however, before this state is obtained. The only discoverable change histologically is in the islets. They show varying degrees of involvement ranging from depletion of the beta cells to complete disappearance of the islets and their replacement by hyalin tissue. These animals differ from de-pancreatized dogs in several respects. They can survive for long periods of time without insulin.

Repeated and prolonged injections with thyroid hormone damage the islets similarly. The histologic injury obtained, however, is less severe than that induced by anterior pituitary extracts or by alloxan. A permanent diabetic state can be produced in this manner only if a portion of the pancreas has first been removed.

It has been claimed that ingestion of excessively large amounts of carbohydrates over a long period of time will first stimulate and finally exhaust the islet cells leading to their permanent destruction. This is an extremely difficult method with which to produce experimental diabetes mellitus.

Another method is by the use of alloxan, and this is the one employed by Dr. Naidoff and his co-workers for their experiments. Alloxan is a specific poison for the beta cells of the islets.

It is important to realize that, in a human with diabetes mellitus, gross microscopic changes in the pancreas are rare. Edema of the beta cells has been demonstrated. However, usually no anatomic site for the clinical diabetes mellitus can be found. This fact must be kept in mind in evaluating any ocular changes produced by experimental diabetes mellitus.

Of the several experimental methods for the production of diabetes mellitus, a few have been employed for the production of experimental cataracts. Dr. Naidoff and his group have used the method of alloxan. Foglia and Cramer employed pancreatectomy in 1944, and were able to develop cataracts in all rats from whom 95 percent of the pancreas had been removed. These cataracts developed within 50 days. When 80 percent of the pancreas had been removed the cataracts developed more slowly taking approximately 200 days.

There are several possible mechanisms by which cataracts may be formed in diabetes mellitus. They may result from hyperglycemia. The observations of Dr. Naidoff and his co-workers tend to support this mechanism.

Patterson, in 1944, was able to produce cataracts experimentally by the intravenous injection of dehydroascorbic acid and related compounds in rats. A hyperglycemia is produced with these agents through a mechanism of partial saturation of the renal-tubule reabsorption pathway for glucose.

Patterson was able to correlate the onset of the cataracts with the severity of the hyperglycemia. When the blood sugar was high, the cataracts developed in eight to 10 weeks; when the blood sugar was lower, the cataracts were slower to develop and were often unilateral.

Another possible mechanism is interference with enzyme metabolism within the lens.

Bellows found that the glutathione concentration of the lens was reduced within 72 hours after the administration of alloxan. Dr. Naidoff and his co-workers have demonstrated that the earliest changes in the lens could be detected within 24 hours after the administration of alloxan. Thus, since glutathione depletion does not occur for three days, it would appear that this is not the fundamental fault, since the cataractous changes precede the glutathione depletion.

Other mechanisms must be considered for the production of the cataracts. Edema and degeneration of the ciliary epithelium, as the result of hyperglycemia, perhaps might lead to lens changes on a nutritive basis. Alterations in the permeability of the capsule, hydration of the lens by alterations in osmotic pressure relationship, and hormonal imbalance are all possible routes by which the lens vacuoles, fissures, and opacities develop.

Bellows and Shock (1950) noted that the earliest lens changes took place in the periphery and consisted of small vacuoles. Dr. Naidoff and his co-workers were able to confirm this site of initial change. However, the experimental reports tonight showed the onset to occur within 24 hours.

I was impressed with the fact that Dr. Naidoff, Dr. Pincus, and Dr. Town could employ a method that has been available for many years, and has been used repeatedly by many workers for the production of cataracts, and by painstaking, careful, and thorough research add something to our knowledge of the formation of experimental diabetic cataracts. I look forward with great interest and anticipation to the continuation of these studies.

Dr. I. S. Tassman: The results of experiments on animals of this kind do not always indicate what occurs clinically. There are one or two questions that arise with regard to the conclusions and the conduct of the experiment. I wonder whether ophthalmoscopic examination of the fundus of the eye was made in those eyes in which it was possible to do so, and whether or not it was possible to recognize the presence of any retinopathy. There are certain factors no doubt present in the diabetic state in humans which do not enter into a problem of this condition in rabbits.

Becker and Friedenwald at the present time are conducting studies at the Wilmer Institute with regard to the occurrence of retinopathy in alloxan diabetic rabbits, and the effect of administration of corticotropin in diabetic rabbits. They have, up until the present time, I believe, found that retinopathy occurs in the diabetic rabbits who have also received corticotropin. The alloxan diabetic rabbits who have not received the corticotropin showed no retinopathy, I believe, and in the control group or nondiabetic rabbit the retinopathy was absent.

If a complete cataract develops in these cases, in the short period of time that was mentioned, it would prevent an ophthalmoscopic examination of the fundus, but in the other cases in which it was possible to examine the fundus, I wonder whether or not any evidence of retinopathy was present.

As Dr. Leopold stated, there is decrease and loss of glutathione in the lens with the development of cataract, and in the mature cataract, even in humans, it has been found that glutathione is absent or almost entirely absent. However, I don't think Bellows should be given the credit for this. This was demonstrated long before by Adams in England, and at about the same time by someone in this country whose name escapes me at the moment.

Dr. Joseph Waldman: I merely wanted to know about the protecting action of Bal against alloxan, and Dr. Pincus told me that he didn't go into that phase of it.

Dr. Irving H. Leopold: Dr. Bellows was not the first to describe the loss of glutathione from the lens which developed a cataract from any toxic agent. However, he did observe the loss of glutathione associated with the development of cataracts following the

administration of alloxan, and it is this fact which I mentioned tonight.

Dr. Waldman asked concerning the possibility of Bal being used to prevent or clear up the opacities of the lens induced by alloxan diabetes. Lazarow demonstrated, in 1944, that the use of glutathione could prevent the damage to the islet cells by alloxan.

Buschke attempted to prevent, by means of Bal, the development of cataracts following the use of naphthalene and galactose. Both of these toxic agents, naphthalene and galactose, cause a disappearance of glutathione from the lens. Buschke was unable to influence cataracts which had already started by employing Bal. Cysteine is a constituent of glutathione which contains the all-important sulfhydryl radical. Others have shown that, if cysteine were employed prior to the development of galactose or naphthalene cataracts, the lens damage would be somewhat reduced.

Dr. I. J. Pincus: I agree heartily with Dr. Leopold that the study of the eye in relation to metabolic disturbances is an extremely fascinating field. We have studied a number of factors in our animals, and attempted to correlate these with the eye changes.

Lipid analyses have been made of blood and liver and, although these were related to the blood-sugar level in a general way, there were exceptions which were quite striking, and it was apparent that only an indirect relationship existed between the lens changes and the blood or liver lipids.

Our animals were well studied in regard to their "diabetic" state. I think it of some interest that some of our animals have lived as long as a year after the administration of alloxan with blood-sugar levels as high as 600 mg./100 cc.; these animals appeared well nourished, frequently gained weight, and seemed quite well. Despite the fact that insulin was not administered to any of these animals, except for one small group, not one showed chemical or clinical evidence of acidosis.

The alloxan-diabetic animal does not resemble the usual diabetic patient, and this is particularly true of the rabbit. The only factors with which the eye changes were directly related were the blood-sugar levels, urinary glucose excretions, and the duration of the "diabetes."

Since the report from the Hopkins group we, too, have been interested in the retinal changes which they reported after ACTH. A group of rabbits severely diabetic following the injection of alloxan were given varying dosages of cortisone. These animals promptly became very ill, they lost weight, and several died. The remainder were killed and a thorough study of the kidneys, heart and retinas, as well as other tissues, showed no evidence of a vascular disturbance. Whether ACTH will have other effects we do not know.

Our studies on the lens are continuing, and we hope to have more to report in the near future.

Dr. David Naidoff: Although most investigators of this problem have stressed the similarity of alloxan diabetes to human diabetes, we have not made any attempt to correlate the two conditions in this study.

I should have mentioned that the fundi of all the animals were examined routinely, but we were never able to demonstrate any diabetic retinopathy. We also tried to produce diabetic retinopathy in alloxan-diabetic animals by injecting them with cortisone. Our rabbits immediately developed more severe diabetes, and died very soon thereafter.

Again, no retinopathy was observed, but it should be stated that, in these rabbits, lens changes were severe enough to prevent a thorough examination of the fundus, and that the rabbits used had been severely diabetic for a long time previous to the cortisone injection.

I would like to thank Dr. Leopold, Dr. Tassman, and Dr. Pincus for their very interesting and informative discussions.

M. Luther Kauffman,
Clerk.

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AN OCULIST WITHOUT A PATIENT

The circumstances surrounding the attempt of Sr. Arthur Conan Doyle to specialize in diseases of the eye led not only to his decision to abandon his medical career, but also to a greater fame as creator of the incomparable Sherlock Holmes.

Following the birth of his daughter in January, 1889, a new feeling of parental responsibility came upon Dr. Doyle. He became aware, in spite of his steadily increasing literary success, that his life as a provincial practitioner of medicine was leading down the narrow path of mediocrity. He was falling, as he put it, into a rut. He often considered concentrating his efforts in the field of literature but he was reluctant to give up his medical profession.

On sudden impulse, the end of October, 1889, Dr. Doyle decided to make a hurried trip to Berlin to attend a lecture and demonstration on an alleged cure of tuberculosis by the famous Dr. Koch. His chance companion on this journey was a Dr. Malcom Morris of London.

Dr. Morris had also been a provincial physician but had come to London and established a successful practice as a skin specialist in Harley Street. After long talks and exchange of confidences, Morris urged Doyle to give up his practice in Southsea and seek a broader scope for his talents in London. Morris suggested that he start as a practitioner of some specialty, after some study on the continent.

On learning that Doyle had been interested in eye work, and had been studying optics, doing refractions and ordering glasses in the Portsmouth Eye Hospital under Dr. Vernon Ford, Morris suggested that Doyle go to Vienna for six months, come back and start in London. "Thus you will have a nice clean life with plenty of leisure for your literature."

It was not difficult to pack up and depart from Southsea. After eight years as a general provincial physician, his practice had dwindled, with so much of his time spent in writing. Leaving his daughter with his wife's mother, he and his wife arrived in Vienna in a snow storm, December, 1890.

In his autobiography, Dr. Doyle states that his four months attending eye lectures at the Krankenhaus were somewhat of a disappointment; he felt he would have learned far more if he had stayed in London. He possessed fair knowledge of conversational German, having spent a year in a boy's school in Feldkirch in western Austria during the earlier years of his schooling, yet he found it difficult to follow accurately the lectures filled with technical terms.

Vienna in the year 1891 was at the height of its medical popularity. In that year the two University Eye Clinics were headed by the famous Dr. Ernst Fuchs and Dr. K. Stellwag von Carion.

The Viennese era of scientific ophthalmology, starting under the teachings of Prof. Ferdinand Arlt, attracted to Vienna students and patients from all over the world. Ernst Fuchs was Arlt's assistant from 1876 to 1880 and succeeded to the professorship in 1885, following Professor Jaeger. Fuchs was a man of extraordinarily striking personality and scientific attainment and achievements; his lucid teachings, clinical and pathologic investigations, and writings gave to Vienna fame not to be denied.

Dr. Doyle does not mention his teachers in his autobiography. Professor Fuchs was a brilliant linguist but, according to his distinguished son, Dr. Adalbert Fuchs, he did not give lectures in English. In all probability Doyle attended the lectures of both professors. Von Carion's contributions to ophthalmology include a *Textbook of Practical Ophthalmology*, a monograph on failure of accommodation, investigations of anomalies of refraction, and research on glaucoma and polarization of light in the human eye. Doyle may have been attracted to von Carion because of his interest in the field of refraction.

After visiting with Landolt in Paris for a few days, Dr. Doyle returned to London in March, 1891. Landolt was the most famous French oculist at that time. Doyle considered the winter wasted in spite of the increased prestige of "having studied in Vienna."

In selecting his office in London, he sought one close to Harley Street whereby the outstanding oculists would possibly send him patients for refraction. He thought that they disliked to spend time in working out the refractive error, "which in some cases of astigmatism take a long time to adjust when done by retinoscopy" and might send patients to him. He considered himself capable of the work and liked it.

He found quarters at 2 Devonshire Place, at the top of Wimpole Street and close to

famous Harley Street. For 120 pounds a year he obtained a front consulting room and part use of a waiting room. He humorously remarked that he was soon to find them both waiting rooms.

Not a single patient crossed his threshold. However, the quiet hours in the consulting room were ideal for reflection and literary effort and resulted in some of his best Sherlock Holmes' adventures. Between the middle of April, a month after his return to London, and August, 1891, he finished six short stories. Between the time the first adventure, *Scandal in Bohemia,* appeared in the July, 1891, number of the *Strand,* and the sixth, *The Man with the Twisted Lip* in the December, 1891, issue, Sherlock Holmes and his creator rose to the eminence of unfading literary renown.

He kept his professional office open for five months, until August, 1891. Following a severe attack of influenza, he decided to cast aside his medical career and live entirely by his writing.

Francis H. McGovern.

THE NUMBERED INTERNATIONAL CONGRESSES OF OPHTHALMOLOGY

The International Council of Ophthalmology was formed on July 14, 1927. Not less than 43 delegates from 24 different nations met in Schveningen and drafted the statutes for the future international congresses. To strengthen the basis of international intercourse and to secure its continuity, independently of political vicissitudes, Gonin had proposed the formation of an International Association of Ophthalmologists with the individual ophthalmologists of different countries as members. Treacher Collins, on the other hand, seconded by von der Hoeve, had suggesteed a Federation of Ophthalmological Societies with the national societies as members. The latter suggestion was accepted and, at the XIV Congress in Madrid in 1933, the federation was formed and its statutes accepted. By these statutes the International Council was made the administrative organ of the federation, retaining at the same time the final responsibility for the congresses. Provision was also made for an electing body of delegates from the member societies and an executive committee to operate for the council.

At its session in Cairo in 1937, the Association for Prevention of Blindness was affiliated with the International Congress of Ophthalmology. The International Organization Against Trachoma, founded at that meeting, was also affiliated and these two organizations became auxiliaries of the congress. The presidents of these two organizations were made members ex officio of the council. These organizations have since then co-operated with the federation, synchronizing the sessions of their council and delegation with those of the latter.

While endeavors were made to hold congresses every four years, there were gaps from cancellation because of international political strife. Here is a list of planned congresses as held:

I	1857	Brussels
II	1860 1862	Paris
III	1867	Paris
IV	1871	London
V	1876	New York
VI	1880	Milan
VII	1880	Heidelberg
VIII	1894	Edinburgh
IX	1900	Utrecht
X	1904	Lucerne
XI	1909	Naples
XII	1914	St. Petersburg
XIII	1929	Amsterdam
XIV	1933	Madrid
XV	1937	Cairo
XVI	1950	London

Since its foundation in 1927, the International Council has met at least once a year. Together with the local committees, it has had four congresses and selected Montreal

and New York for the XVII Congress in 1954.

For several years the congresses were deferred because of strained international diplomatic relations in Europe. Following the Cairo meeting in 1937, a movement was inaugurated by Harry S. Gradle, Moacyr E. Alvaro, and Conrad Berens to organize a Pan-American Congress of Ophthalmology and, sponsored and abetted by the American Academy of Ophthalmology and Otolaryngology, a full-blown scientific meeting was held in Cleveland, Ohio, in 1940, and a permanent organization established. The International Council at once recognized the Pan-American Association and concluded an agreement about synchronizing the International and Pan-American Congresses so that a congress of either kind would take place every other year.

The Pan-American Congresses have been held according to plan:

I	Cleveland, Ohio	October, 1940
II	Montevideo, Uruguay	November, 1945
III	Havana, Cuba	January, 1948
IV	Mexico City	January, 1952

The V Pan-American Congress will be held in Santiago, Chile, January, 1956. The time and place of the XVIII International Congress of Ophthalmology will be determined by the council at their next plenary session in Montreal in September.

William L. Benedict.

OBITUARY

EUGENE WOLFF
(1896-1954)

Eugene Wolff, F.R.C.S., ophthalmic surgeon to the Royal Northern Hospital and surgeon to Moorfields, Westminster, and the Central Eye Hospital, died in London on February 25th. He was born at Oudtshoorn, Cape Province, South Africa, but came as a boy to University College School, Hampstead, from which he went on to University College, London, and then to University College Hospital, where he was awarded the Lister Medal for clinical surgery in 1918. In the same year he was graduated from London University with the degrees M.B., B.S.

Following a year's service as captain in the South African Medical Corps, Mr. Wolff returned to University College in 1919 and, for the next eight years, pursued studies which enabled him to make valuable contributions to the anatomy and pathology of the eye. Meanwhile, clinical work was not neglected and he became house surgeon and afterward ophthalmic registrar to the late Percy Flemming and Sir John Parsons at University College Hospital, as well as chief clinical assistant at Moorfields Eye Hospital. In 1927 he became a Fellow of the Royal College of Surgeons, England.

During most of his years as demonstrator of anatomy at University College, Mr. Wolff found time to lecture on this subject in the Slade School of Art and to write his first book, *Anatomy for Artists* (1925), which is now in its third edition. There followed, in addition to a number of excellent papers, three fine books, *Anatomy of the Eye and Orbit* (1933), *Pathology of the Eye* (1934), and *Diseases of the Eye* (1937). All of these books are beautifully illustrated with his own drawings. The popularity and value of these books are shown by each going into several editions.

Many American ophthalmologists serving in the European Theater during World War II remember Eugene Wolff with much gratitude and affection for his many kindnesses to them during this trying period.

In 1945, Mr. Wolff was elected a vice president of the Ophthalmological Society of the United Kingdom. He was also an honorary member of the Ophthalmological Societies of Belgium and of Greece, and a member of the Section of Ophthalmology of the Royal Society of Medicine and the Section of Ophthalmology of the British Medical Association.

In 1923, Mr. Wolff was married to Lydia Abravanel of France, who with their daughter, Audrey, survives him.

CORRESPONDENCE

Standard visual acuity chart

Editor,
American Journal of Ophthalmology:

The internationally accepted nomenclature of visual acuity is another of those things about which not much is being done although (especially in years preceding international gatherings of ophthalmologists) many people talk about it. The American Committee on Optics and Visual Physiology proves to be a laudable exception and the subcommittee it appointed did a masterful piece of work toward clarifying the underlying issues. Everyone interested in this important subject must have enjoyed the report published by the chairman of this subcommittee, Kenneth N. Ogle, in the July, 1953, issue of The Journal.

Before making any specific recommendations on this topic, may I be permitted to repeat some of the conclusions at which the committee arrived. One will, no question, fully agree with almost all of them.

1. Test charts are just as important "to determine the effect of certain ocular diseases" as they are "to assist in the determination of the refractive correction." Thus, although visual acuity charts are used primarily to determine refractive errors, "the design should be such that determination of visual acuity itself could be made with reasonable accuracy."

2. The Snellen notations, such as 20/20, 5/6, or 14/16 are admissible only if the visual-acuity determinations have been made at 20 feet, 5 meters, or 14 inches, respectively.

3. As to the design of test letters and test charts, the majority of the committee feel that a *geometric progression* in the size of letters of successive lines is desirable.

4. A geometric progression in which the ratio of the size of one line of letters to that of the preceding line is about 1.25 is satisfactory.

There are, I may add, 10, obviously equal, steps from 6/6 (or 5/5 or 20/20) to 6/60 (or 5/50 or 20/200) in a geometrically progressive system of this ratio, while there are only nine, and unequal, steps in the "international" decimal system. This, without question, assures the greater accuracy of the former.

7. The decimal system as well as the Snellen notation has the basic assumption that 1.00 represents "normal" vision. "It is unfortunate that the term 20/20 and its designation in decimal form has become a fetish in the popular mind."

8. The underlying factor in all visual-acuity testing is the *resolving power of the eye*. The letters are designed to measure, so far as possible, that resolution. *Visual acuity should therefore be stated in terms of the quantity that is actually measured,* that is, in terms of "the visual angle of resolution."

Though the subcommittee, for some reason, has not cared to be more specific in its recommendations, it obviously meant that visual acuity should be designated in terms of, say, "2 minutes" instead of 20/40, or 5/10, if the minimum visual angle of resolution happens to be two minutes. Should this recommendation be accepted by the coming congress, we would, I am quite sure, soon learn the new way of expressing ourselves. Reporting, for example, on the loss of vision in the course of a case of glaucoma, we would not say any more that the patient's vision deteriorated from 5/4 to 5/10, but that it dropped from 45 seconds to 2 minutes. We would possibly continue to add that visual acuity was measured with a chart set at, say, five-meters distance, just as we usually state that the tonometer weight was, say, 7.5, even if we report that intraocular pressure was 32 mm. Hg.

Whether or not we would continue to use

two numbers to express one value, this very point makes it obvious that the international committee which should finally issue such charts would have to issue a series of sets—different sets for those who test their patients at 5 m., 6 m., 20 feet, and so forth.

Some "standard" set just would not suffice because even if that international body would decide for, say, 6 m. as the "standard" testing distance for its approved visual-acuity chart, I, for one, would still continue to have my chart set at 5 m. My examining room just is not long enough and I don't care to use a mirror. Different test charts would thus be needed, for 4, 5, 6 m., and so forth, not to speak of 20 feet.

And, if the committee feels that a report specifying the patient's visual acuity as 20/40 is accurate only if this acuity has been tested with a chart at 20 feet, one cannot see how, and why, the statement "2 minutes" for the same visual acuity should be more accurate, or even equally accurate, if it does not contain a second number specifying the test distance.

The question that necessarily arises at this point is whether or not we would gain anything by such change in nomenclature and—at least in this correspondent's mind—it is doubtful that we would. On the other hand, nothing is lost either in accuracy of measurement or in international communicability of results if, instead of the smallest angles of resolution (45 seconds and 2 minutes in the just-quoted case) one continues to refer to the distance at which those 45-seconds letters, or 2-minutes letters subtend one minute of arc—and that, after all, is what we have been doing all along.

Rightly or wrongly (and this correspondent feels quite rightly), the notion of "one minute" plays such a significant rôle in the history of determining visual acuity that one hesitates to give it up completely.

Visual acuity of 5/10 does not mean, and, at least for me, does not even suggest an efficiency of 0.5. It simply means that the subject was tested at 5 m. and that, at the test distance, he only read those letters, details of which subtend one minute of arc at 10 meters. But if 20/20 or 5/5, in the committee's opinion, still suggest the false "normality" of 1.00 or 100 percent, then the best thing to do is to drop the numerator of these *apparent* fractions.

In fact, the visual acuity of the just-quoted case of glaucoma could just as satisfactorily be described by stating that the patient's visual acuity dropped from 4 m. to 10 m. or simply from 4 to 10. Adding that measurements were done at 5-m. distance, our statement would be as correct as the committee could wish. Our statement would emphasize our awareness of the fact that what is involved in our report are visual angles and this is what the subcommittee wants our statements to emphasize.

Besides, we would not confuse the uninitiated or the conservative. Both would immediately realize that what we now call a visual acuity of 4 or 10, taken at 5 m., is the same visual acuity which was previously called 5/4 or 5/10.

In fact, the visual acuity chart of Walter B. Lancaster, published by the American Optical Company, carries just such designation. The numbers 4 or 5 or 64, and so forth, next to the different lines of letters on the Lancaster chart, mean that the letters in those lines subtend a visual angle of one minute per detail, at the distance of 4 or 5 or 64, and so forth, meters. There are no fractions, no decimals on this chart. Any suggestions of normal values or percentages is thus impossible.

But this is only a minor feature of this excellent chart. The incorporation of the geometric sequence 1.25, recommended by the subcommittee, makes the Lancaster chart *the best of all so far published charts*, the adaptation of which this correspondent highly recommends. It would immediately eliminate the need for the publication of different sets of charts.

As emphasized already, any of the present Snellen charts are, and any chart carrying the feature "one minute," and so forth, as recommended by the subcommittee, would necessarily be, correct only *when used at one certain and constant* distance.

The Lancaster chart is equally accurate whatever the testing distance. It can be used at the bedside, at, say, 2.5 m. At this distance it is obviously the letter corresponding to the Lancaster notation 2.5 which *is the* 20/20 letter, the letter details of which subtend "one minute"; and the letter which carries the notation 25 corresponds to 20/200. It is equally usable for research work where greater test distances are preferable for greater accuracy.

Stenström, in his monumental study, placed reading charts at 10 m. Obviously, at 10-m. distance, it is the letter with the Lancaster notation "10" details of which subtend "one minute" angles at the eye.

If one uses the Lancaster chart routinely in the office, one quite naturally keeps the chart at some constant distance; all that is necessary in this case is to jot down one number. If one's test chart is at 5 m., one will in the above given example have a record of visual acuity dropping from 4 to 10. And should this very same patient now be tested in consultation in another office where the Lancaster chart is at 6.25 m. (about 20 feet), one could confidently expect that there he will read the 12.5 line. (Originally, of course, he would have read the 5 line in this latter office.)

The geometric sequence of the Lancaster chart is mathematically not quite correct and this is another of its great practical advantages. Taking any step as the starting point, one will find that the letter of the *next* step is about (not exactly) 25 percent larger; that the *fourth* step is always 100 percent larger than the starting point; and that the *third* step is about 20 percent smaller than the fourth.

Thus one finds "groups" of four, like 4, 5, 6.25, 8; or 5, 6.25, 8, 10; or 8, 10, 12.50, 16, and so forth. Two groups together make seven steps and the seventh step is four times the starting step. The 10th step is always about 10 times larger than the basic step, thus the 10th step from 5 is 50, from 6.25 is 64, and so forth. Since the chart in my office is 5 m. from the patient, my record of the visual acuity that is now generally designated as 20/200 is 50.

Continental ophthalmologists should have little trouble getting used to Lancaster's designation since they use a metric system, a 5- or 6-m. testing distance. (Actually, they should set the chart either at 5 or at 6.25 m.) But American ophthalmologists or optometrists finding themselves in need to communicate with a fellow clinician used to the "traditional" system would not experience great inconvenience either. Assuming a testing distance of 5 m., 5 means 20/20; 10 means 20/40; 50 means 20/200, and so forth. Obviously, all that is needed is multiplication of the Lancaster number by four to get the correct denominator.

However, there are certain limitations to the Lancaster chart, too. Registering its single numbers of designation is still not sufficient; one still has to communicate, or at least keep in mind, the distance at which the chart was read. The notation "5" might mean 20/20 for the one clinician who uses the chart at 5-m. distance; 20/15 for another whose chart is at 6.25 m.; and even 20/10 for the research man with the 10-m. room.

Testing visual acuity is, moreover, testing a performance and *it is just not quite logical to tag a smaller number on a better performance.* An IQ of 120 is "better" than one of 90 and a visual acuity of 20/20 (the only good point in looking at it as a fraction!) is "better" than one of 20/200.

Besides, a performance should have a unit, a name of its own; it should not be expressed in other terms—be they angles or distances. One cannot see why, with all the "mach"s

and "bel"s and "hertz"s we hear of lately, we should not distinguish our specialty by simply stating that the performance called visual acuity is to be expressed in a special unit, in *"snellen."*

A person whose angle of *minimum separabile* is "one minute" should be talked about as having a visual acuity of 50, or 60, or 100 snellen. A person with better resolving power should be said to have a visual acuity of *more snellen*. And if resolving power is poorer, the number which characterizes this performance should be *smaller*.

The actual number is a matter of choice. One would think of 100 as first choice because it is a round figure, were it not for the subcommittee's strong feeling against 1.00 or 100 which suggest normal, or 100 percent, or best. This feeling is probably wrong. I hardly ever fail to make some youngsters (and their parents) happy by telling them that their visual acuity is 120 percent or better than average, once they read the line under the 5 line of my Lancaster chart.

The designation "60 snellen" for 20/20 has also much to recommend it. The number 60 reminds one of the 60 seconds which details of the 20/20 letter subtend, and of the 60 oxyopters in Blaskovicz's very beautiful system. But possibly 50 snellen is the best choice for 20/20 vision, by reason of its easy memorization.

Accepting this choice, one will say that a patient reading Lancaster's 5 at 5 m. sees 50 snellen, and one reading Lancaster's 50—the 20/200 of the traditional nomenclature—sees 5 snellen. In fact, to the already available Lancaster charts could easily be added some extra numbers so as to carry *both* notations:

(1) The notation of distance to which we have become conditioned through almost a century; and (2) the notation of a performance measured in its own units. Assuming that the chart is at 5 m. one will find that the following relationship exists:

LANCASTER NOTATION	SNELLEN NOTATION	"SNELLEN"
2	20/10	100
3.125	20/12	80
4	20/16	64
5	20/20	50
6.25	20/25	40
8	20/32	32
10	20/40	25
12.5	20/50	20
16	20/64	16
20	20/80	12.5
25	20/100	10
32	20/130	8
40	20/160	6.25
50	20/200	5
64	20/250	4
80	20/300	3.12
100	20/400	2

One cannot help nowadays that the nonmedical public partake in discussion of matters relating to health and performance. A youngster, an anxious parent, a candidate for a job or a special branch of service, will want to "know what his vision is" and will always, and sometimes inaccurately, use terms of the common medical vernacular.

Mothers come to the office reporting that the school nurse said their child's visual acuity is 40.

"Is this good or bad?"—they continue. "The neighbor's daughter sees 20. Is that better or worse?"

Why not have the numbers right? Let the mother report that the nurse said the child's vision is 25, or still better, 25 snellen. If we can improve it to 50, we shall make her satisfied because 50 is more than 25 and we can tell her that it is "normal." The public wants to know what "normal" is, even if we, among ourelves, only call it "average." If we can improve it to 64 or 80, we shall tell her her child's vision is "superior." The objectionable term 1.00 or 100 percent will thus be avoided.

Since, theoretically, any line of the Lancaster chart is a potential 20/20 line, the

"snellen" notation can be added according to the examiner's choice of testing distance. All that is necessary is to shift the third column of the table relative to the first. If visual acuity is being tested at 6.25 m. (about 20 feet), 50 snellen of the third column has to be shifted to the level of 6.25. In this case one will find that Lancaster's 16 equals our 20 snellen, his 25 our 12.5 snellen, his 64 our 5 snellen or the traditional 20/200, and so forth. The sequence of snellens will remain the same and without any further data communicable. A person having a visual acuity of 50 snellen will show the same visual acuity in any office, whatever the distance of the chart.

Once more, the feature of groups might be emphasized. Improvement of vision (or decrease) by the same number of lines always means the same gain (or loss) wherever one starts. There are three steps from 50 snellen to half of it, to 25 snellen; again three steps from 25 snellen to half of it, 12.5 snellen, and so forth. And there are always 10 ample (and equal) steps from "average" (not "normal") to the ominous 20/200 of "industrial blindness."

I plan to make a formal motion at the next international meeting to the effect that (1) the Lancaster chart published by the American Optical Company be accepted as the standard visual acuity chart; and (2) that the "snellen" be accepted as the international unit of visual acuity.

As meetings go nowadays, with their crowded schedules and limited discussion time, these motions will again be delegated to a committee and no action taken.

THE JOURNAL could do a great service in expediting this matter by (1) permitting other journals to reprint part, or all, of this letter; and (2) by encouraging other letters to you. By the time of the September gathering, public opinion could be sampled well enough for definite action.

I expect that there will be discussion on my first suggestion. After all, the Lancaster chart is not the best possible chart so far as selection and form of letters, serifs, thickness of stroke, and so forth are concerned. But there could hardly be any dissent on my second suggestion.

The word "snellen" is easily spelled and equally pronounced in every language and the name "Snellen" is known and has been known to millions of ophthalmologists, optometrists, nurses, dispensing opticians, public health officials all over the world and his chart hangs in the office of every practicing physician.

(Signed) Arthur Linksz, New York.

BOOK REVIEWS

PHYSIOLOGY OF THE EYE. VOLUME 2: VISION. By Arthur Linksz, M.D., New York, Grune & Stratton, 1952. 869 pages, 248 figures, bibliography, author and subject indices. Price: $19.00.

To ophthalmologists interested in action rather than thought, the field of the psychophysiology of vision is largely *terra incognita*. Now that this fine monograph by Linksz is available the American Board of Ophthalmic Examinations may do well to place more emphasis on the subject than hitherto. Some time ago a candidate for the Board questioned by an examiner about the horopter replied that though he had seen the instrument he had never used it.

The lack of parallelism between objective stimulus and subjective interpretation has created historic conflicts such as that of Goethe versus Newton due to overemphasis of one phase or the other of this duality. Though Linksz has a flair for subjectivism and has been thinking through the problems of vision from this standpoint for over 15 years, his approach is well balanced and the diverse contributions of Ames and Lancaster are analyzed with equal reverence. The board implications of the subject range from

philosophy—the problem of knowing—to the history of art, and the various challenges are fairly met.

The description of size lenses is too diffuse because of an obvious effort to avoid mathematics. The introduction of the formula of a zero-power lens would certainly have simplified the explanation. In color vision no other ophthalmologic text is so detailed and up to date in the data presented. In discussing binocular vision, Linksz considers fusion and divergence as purely reflex processes and finds no need to assume special centers for these functions. "A name given to a concept does not turn that concept into a fact."

Through oversight, the review of this important book has been delayed until now. It is hoped, however, that this late notice may give a fresh stimulus to this significant work.

James E. Lebensohn.

Diseases of the Retina. By Herman Elwyn, M.D. Philadelphia, The Blakiston Company, 1953. Second edition, revised and augmented. 697 pages, 243 illustrations, 20 in color, index. Price. $12.00.

The second edition of this excellent work, coming so soon after the appearance of the first edition, is an index of its popularity and usefulness, not only to ophthalmologists but also to general practitioners, internists, neurologists, and advanced students in ophthalmology.

In addition much new knowledge concerning many diseases that affect the retina has been acquired since the first edition, and the author has found it necessary to revise some of the chapters (for example, essential hypertension, diabetic retinopathy) and add others (for example, retrolental fibroplasia, ocular tuberculosis, sarcoidosis, toxoplasmosis). There are new illustrations, some in color, and the printing is exceptionally well done.

This is an exceedingly useful and valuable book, well annotated, with satisfactory references and index. It deserves the popularity given its predecessor.

Derrick Vail.

The Floating Contact Lens. By Dr. Otto Knüsel. Basel, S. Karger, 1953. 63 pages, 36 illustrations, some in color. Bibliography. Price: s Fr. 7.30.

This small monograph on the "fluidless contact lens" acquaints the European ophthalmologist and technician with the Tuohy lens. After a brief historical introduction, there is a presentation of pertinent mathematical and optical facts. The author points out that Bürki's simplified formula is not quite satisfactory because it neglects the effect of the "water lens."

Knüsel first performs an objective and subjective refraction and expresses any astigmatism present in the form of a minus cylinder. The minus cylinder can be disregarded. He determines the value of the resulting sphere for a position at the apex of the cornea. Next, he inserts an afocal contact lens that fits the cornea well, repeats the refraction, and calculates the new value for the vertex of the cornea. The difference between these two values indicates the refractive power of the concave water lens. The value determined for the anterior principal point is incorporated into the definitive lens.

Two curves, one for the phakic and one for the aphakic eye, give an excellent graphic demonstration of the difference in magnification of the retinal image due to a spectacle and corneal lens respectively.

Knüsel suggests that patients who might benefit from telescopic lenses should wear a strong myopic corneal lens as the ocular with an ordinary plus lens in a frame as the objective. Such an arrangement is less conspicuous and more comfortable than the customary telescopic glasses. At the same time, it

affords a wider visual field. This principle is, of course, not an original one.

In the chapter on fitting the lens, Knüsel describes the technique of Tuohy, Anderson, and Salvatori. He finds it easiest to insert the lens by means of a suction cup. He brings the lens within one or two millimeters of the cornea and exerts a slight pressure on the cup causing the light lens to be blown against the cornea. This is less irritating to the eye than the customary procedure.

Note the author's patience in gradually increasing the period in which the lens should be worn. For an entire week, it is not left in place for more than two hours. After one week, the duration is increased one-half hour every day. He takes great pains in selecting not only the proper radius but also the right diameter for the lens. A lens with a diameter that is too small and moves too freely could easily create the false impression that the radius is too big.

Though Anderson thinks that the lens can touch the cornea and yet be tolerated, the author believes that the faint glow of an Argon lamp is too weak to render the fluorescein film visible. However, with stronger light, a thin fluorescein film can always be demonstrated if the lens is tolerated.

Salvatori prefers lenses with a radius shorter than that of the cornea. In his opinion that is the best way to avoid corneal erosions.

Knüsel uses a one percent aqueous solution of Trypan blue as a vital stain in addition to fluorescein. Whereas, the latter does not stain the cells themselves but the interstices, the former actually stains cells and allows for much finer differentiation of any damage—according to the amount of dye accepted. On that basis, the postulate which rules out a contact lens that *touches* the cornea is changed to a postulate which rules out a contact lens that *damages* the cornea: The pressure of a well-fitting lens is no greater than that of the lids. A well-fitting lens can be tolerated for 12 hours or more without provoking fluorescein—positive points on the cornea; it floats freely over the corneal surface.

As an additional aid for evaluation of a proper fit, Knüsel instills a drop of liquid paraffin, and adds Sudan Red. The size of the resulting droplets and their distribution enables one to judge the adequacy of the lens. At first, winking of the lids causes a whirl of droplets distributed rather evenly over the entire surface of the cornea. After a while, the droplets are absent from the periphery and remain only over the center of the cornea in a stationary position.

One chapter is devoted to representative cases, including keratoconus, corneal maculae, aphakia, and excessive myopia. Knüsel would consider keratoplasty for corneal opacities only after attempts to improve the visual acuity by means of contact lenses have been unsuccessful.

Being different from most writers whose enthusiasm for their subject carries them away, Knüsel is quite strict in his indications and conclusions. His little brochure should create an important impetus for wider acceptance of the Tuohy lens in Europe. His plea to ophthalmologists to cooperate to a fuller extent with the technician and to familiarize themselves with the few and simple principles pertaining to a perfect fit of the fluidless lens are of general significance.

Stefan Van Wien.

PITUITARY CHROMOPHOBE ADENOMAS: NEUROLOGY, METABOLISM, THERAPY. By John I. Nurnberger, M.D., and Saul R Korey, M.D. New York, Springer Publishing Co., Inc., 1953. 282 pages, clothbound, with illustrations, bibliography, and index. Price: $7.00.

This book may be divided into three sections: the first dealing with the anatomy, embryology, and physiology of the pituitary;

a second dealing with the signs and symptoms of pituitary chromophobe adenoma, and a brief third section covering therapy.

The section on anatomy calls again to our attention the relationship of the pituitary laterally with the cavernous sinuses and their contents, namely the internal carotid artery and the third, fourth, sixth, and three divisions of the trigiminal nerve; superiorly with the optic chiasm and the third ventricle; anteriorly with the anterior communicating artery and the frontal lobes; and posteriorly with the hypothalamus. The familiar diagrams of the four possible relationships of the optic chiasm to the pituitary and the arrangement of the nerve fibers in the chiasm are included. A diagram demonstrating the progression of the visual fields in a typical pituitary lesion (clockwise in the right eye and counterclockwise in the left eye) is helpful.

The second section dealing with signs and symptoms is of particular interest to ophthalmologists. The analysis, based upon 117 critically selected cases of chromophobe adenoma, shows the incidence of visual disturbances as an initial complaint to equal that of all the other introductory symptoms combined; moreover, in those instances where diminution of visual acuity and/or visual field defects were not the primary events, they later accompanied the other presenting complaints.

The following data indicate the distribution of the presenting symptoms: defects in visual acuity and/or fields, 62 cases (47 percent); headache, 26 cases (20 percent); sexual dysfunction, 22 cases (17 percent) with the following symptoms: amenorrhea (15 cases) and impotence (seven cases). The remaining symptoms were distributed among 21 cases (16 percent) and included: extraocular muscle palsies, four cases; hypoadrenalism, three; drowsiness, three; asthenia, two; seizures, two; polydipsia and polyuria, one; gaze palsy, one; hemiparesis, one; visual hallucinations, two.

The visual disorders ranged from a simple diminution of vision to bilateral blindness.

Pupillary size and reactions were specifically recorded in only 21 of the 117 cases. In the majority of these 21 cases the pupil was larger on the same side in which the visual field defect was greatest. The explanation for this is weak.

Extraocular muscles palsies were present in four cases, indicating spread of the adenoma laterally into the cavernous sinus. The third nerve is more frequently affected, followed by the sixth. Conjugate paralysis when present indicates involvement of the frontal lobe. Papilledema was present in seven cases or six percent of the total number of cases.

In 96 of the 117 cases, defects in the visual fields were present: bitemporal hemianopia, 57; blindness in one eye with a temporal defect in the other eye, 19; homonymous hemianopia, three; temporal defect in one eye with a central scotoma in the other eye, four; blindness in one eye with a generalized construction in the opposite eye, four; bilateral contraction of the fields, two; bilateral blindness, four.

Only five percent of these patients who complained of visual disorders or who showed defects in the visual fields failed to show any ophthalmoscopic evidence of optic atrophy.

In the third section dealing with therapy the pituitary adenoma are classified into three groupings: group I, visual acuity in one eye better than 20/50 and in the other better than 20/100 with satisfactory visual fields and no extraocular muscle involvment or evidence of impaired motor power, or obvious psychologic or endocrine defects.

Group II, visual acuity with a minimum of 20/50 in one eye and a minimum of 20/200 in the opposite eye and satisfactory fields with only a minimal or nominal evidence of impaired motor power, psychologic or endocrine disturbances. Group III visual acuity worse than 20/50 in one eye and worse than 20/200 in the opposite eye with

or without satisfactory fields and evidence of major motor, psychologic, and endocrine dysfunction.

The authors feel that Groups I and II should receive radiotherapy accompanied by frequent general and visual examinations. They feel that improvement with roentgen therapy is seen within four to six weeks and little is to be expected if a diminution of pathologic signs is not apparent at that time. Surgery is then advised.

Group III presents a problem in treatment. They feel that a delay of one month during which radiotherapy is attempted will not affect the outcome adversely and if no progress is made during the month of radiotherapy and one additional month thereafter, surgery must be resorted to.

An extensive bibliography and simple index follow. This book should be of particular value as a reference to those ophthalmologists whose practice is closely linked with that of the neurologists and the neurosurgeon.

Joseph E. Alfano.

GLIOMA OF THE RETINA AND PSEUDOGLIOMAS. (In French.) By Marc Adrien Dollfuss and Bertranne Auvert. Paris, Masson et Cie, 1953. 525 pages, 101 illustrations. Price: Not listed.

This impressive well-organized volume deals with one of the most interesting, as well as most distressing, problems in ophthalmology.

A preliminary chapter discusses the terminology of malignant retinal tumors of children. The authors retained the controversial name of glioma because of its precise clinical and etiologic significance, at the same time emphasizing that "glioma" does not refer to the anatomic-pathologic classification of the tumors.

After a short historical review, the main subject, retinal glioma, is introduced in Part I. Successive chapters discuss in systematic manner the hereditary characteristics, the pathologic picture, and the clinical development. The difficulties of the diagnosis and differential diagnosis, especially in children over three years of age, are analyzed. The diagnostic importance of X-ray studies for possible calcifications is stressed. A so-called exploratory paracentesis of the vitreous is thoroughly condemned.

The detailed description of the present-day concepts in the treatment of glioma and a comparatively optimistic outlook is the most outstanding feature of this study. For the monocular affection, enucleation and excision of the orbital part of the optic nerve is the only recognized treatment. Prophylactic irradiation is indicated only in invasion of the optic nerve. In bilateral disease the more extensively affected eye should be removed. The second eye should be removed also, if more than one-third of the retina is involved, if a seeding into the vitreous had taken place, or if the tumor should be exceptionally prominent. In such a situation the necessary irradiation dose would have to be so large that the eye could not survive. In case the tumor in the second eye includes less than one-third of the retina irradiation is indicated.

The second part of this book is devoted to the "pseudogliomas," of which term the authors do not approve. The comprehensive bibliography, arranged according to chapters, refers only to more recent years. For earlier literature the bibliography included in the papers by Wintersteiner, Marwas, and Lagrange are recommended.

Alice R. Deutsch.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

François, J., Rabaey, M., and Vandermeersche, G. **The ultrastructure of the pigment granules of the retinal epithelium.** Ann. d'ocul. **186**:896-900, Oct., 1953.

Studies of pigment granules of the retinal epithelium of man, mammals, amphibians and fish, with the electron microscope, show that: 1. the pigment granules of the retinal epithelium have no internal structure, 2. their surface is irregular and scaly, 3. they have no filamentous prolongations, 4. they have a very pronounced polymorphism, and 5. they are rapidly destroyed by chloramine.

John C. Locke.

Grafflin, A. L., and Corddry, E. G. **Studies of peripheral blood vascular beds in the bulbar conjunctiva of man.** Bull. Johns Hopkins Hosp. **93**:275-289, Nov., 1953.

The painstaking and doubtlessly laborious observations which are reported in this essay are documented with unusually beautiful drawings. In this supplement to earlier studies, selected vascular patterns in normotensive young adults have been analysed and illustrated. The patterns exhibited endless variety and lack recognizable structural and functional units that are built around capillary-like "thoroughfare" channels such as those found by Chambers and Zweifach in other capillary beds. There are, however, numerous arterio-venous communications through which rather large amounts of blood can be delivered into the veins when the capillaries are closed. (10 figures, 11 references)

F. H. Haessler.

Hart, W. M., and Peckham, R. H. **Changes in specific gravity of the growing crystalline lens.** A.M.A. Arch. Ophth. **50**:174-178, Aug., 1953.

After a discussion of chemical changes in lenses with age the authors describe their method of investigation of the specific gravity of the rat lens. They have plotted a curve showing the relationship of the specific gravity and the age of the rat lenses. The curve indicates a maturation process, which reaches its peak 150 days after fertilization. Beyond this point there is no further increase in specific gravity with age. (2 charts, 8 references)

R. W. Danielson.

Koch, C. **Vitreous filaments. Observation on the vitreous of the tuna fish.** Ophthalmologica **126**:58-63, July, 1953.

From the vitreous of a fresh tuna eye threads can be drawn out which upon continued stretching form unstable filaments of microscopic thickness and characteristic structure. (5 figures, 10 references)

Peter C. Kronfeld.

Llombart, A., and Fornes, E. **Characteristics of the sympathetic innervation of the iris.** Arch. Soc. oftal. hispano-am. **13**: 1107-1142, Oct., 1953.

This extensive report of the authors' studies of the sympathetic innervation of the iris of the horse, dog, rabbit and man is preceded by a review of the anatomy of the iris, the technical difficulties which account for the errors of former investigators, and a brief review of the literature. To demonstrate the fine fibers of thin iris sections the authors depigmented the tissue with a concentrated solution of sodium perborate which does not interfere with the staining of nerve fibers. By the use of strong silver stains the fine fibrils could be stained and identified. As the microphotographs show, the distribution of the sympathetic innervation of the iris follows a definite pattern. The fibers enter from the ciliary processes and proceed radially in the stroma of the iris; some terminate in a plexus in the sphincter of the iris, and some end in its retromuscular portion. There are three morphologic components of the sympathetic innervation; the corkscrew fibers, the interstitial cells, and the terminal ramifications derived from these cells. The corkscrew structures in the stroma are believed by the authors to be adaptations to the contractility of the iris, stretching with miosis, and condensing in mydriasis. There are no ganglion cells in the iris; it is believed that the cells described as ganglion cells by some investigators were interstitial cells mistaken for ganglion cells. The interstitial cells of Cajal which form a vast plexus are the most important anatomic unit of this innervation. So far, no one has described the significance of these cells as intermediaries, and their influence on the chromatophores, the dilator muscle, and the posterior pigment epithelium. The chromatophores are innervated by cell contact with processes of the interstitial cells; these processes, touching the walls of numerous cells, influence numerous chromatophores at the same time. The posterior pigment epithelium is innervated in a similar manner. The authors believe that the continuity of the innervation indicates that the sympathetic participates in the metabolism of melanin and influences the function of the chromatophores and the pigment epithelium. The innervation of the sphincter, in the horse at least, has a specific arrangement, which they designate as the "preterminal spiral structure." (25 figures, 11 references)

Ray K. Daily.

Wolff, Eugene. **The so-called medial root of the optic tract is essentially a visual commissure.** Brain **76**:455-456, Nov., 1953.

There is no line of demarcation between the medial and lateral portions of the root of the human optic tract, and there is no real difference in their termination in the lateral geniculate body. Guddens commissure probably does not occur in man. (4 figures, 3 references)

Robert A. Moses.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Fazakas, Alexander. **Comprehensive report of mycotic infections of the eye.** Ophthalmologica **126**:91-109, Aug., 1953.

The well known Hungarian author has, for a good many years, been engaged in

mycologic studies of diseases of the anterior segment of the eye. He now gives a comprehensive report on his findings. Cultures for fungi gave a positive result in 253 (25.5 percent out of 992 individuals with normal eyes and in 820 (36.4 percent) out of 2,210 individuals with diseased eyes. In most of these instances of positive cultures the organism could be identified as to species. In only 35 cases was the ocular disease entirely attributable to the mycotic infection. These were cases of mycotic keratitis, meibomiitis, canaliculitis, blepharitis and conjunctivitis. In another group fungi played the part of a secondary invader and influenced the course of the disease favorably or unfavorably. (19 references)

Peter C. Kronfeld.

Fine, M., and Flocks, M. **Hemagglutination test of Middlebrook and Dubos in ocular tuberculosis.** A.M.A. Arch. Ophth. **50**:163-173, Aug., 1953.

Tests for the diagnosis of ocular tuberculosis are rather unsatisfactory. The skin test is inadequate in some respects and may even be dangerous in tuberculosis of the eye. The authors' study of 101 patients suggests that the Middlebrook-Dubos hemagglutination test may be of considerable value in the diagnosis of tuberculosis of the eye. (2 charts, 3 tables, 12 references) R. W. Danielson.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Fujino, H. **Influence of some surface active agents on the absorption of sulfonamides applied in the cul-de-sac.** Acta Soc. Ophth. Japan **57**:1347-1357, Nov., 1953.

When such active surface agents as aerosol IB, are added to an aqueous suspension of sulfonamides, the absorption of the latter in the cul-de-sac is considerably accelerated. The absorption will be retarded, however, when propylenglycol or carbowax coexist. (6 figures, 1 table, 9 references) Yukihiko Mitsui.

Harris, John E. **Pharmacology and toxicology.** A.M.A. Arch. Ophth. **50**:192-247, Aug., 1953.

In this complete annual review of pharmacology and toxicology the discussions of antibiotics, histamine and cortisone are particularly interesting. (372 references)

R. W. Danielson.

Hofmann, Hans. **Nor-adrenalin in ophthalmology.** Klin. Monatsbl. f. Augenh. **124**:63-76, 1954.

In this careful clinical and pharmacological study the various applications of nor-adrenalin are discussed. It was first used as drops in the conjunctival sac. In 0.05-percent concentration it constricts the conjunctival vessels. The effect is briefer and occurs later than with adrenalin in the same concentration. A 2-percent solution proved to be a good mydriatic (with great individual variations). The mydriasis can easily be overcome with 1-percent pilocarpine. It is useful in iritis with secondary glaucoma. A 2-percent solution can be used as a hemostatic agent. When 1 cc. of a 0.05-percent solution was injected subconjunctivally the mydriasis was maximal. The influence on the intraocular pressure was small and unpredictable. Nor-adrenalin can be added to procaine. The effect is the same as with adrenalin, but shorter. (2 charts, 79 references) Frederick C. Blodi.

Kishimoto, M. **Pituitary removal and vitamin B_1 content in the eye.** Acta Soc. Ophth. Japan **57**:1409-1418, Nov., 1953.

After removal of the pituitary body in the rabbit, there is a change in the permeability of the blood-spinal fluid and the blood-aqueous barrier. A reduction of vi-

tamin B_1 in the spinal fluid and aqueous results. (3 tables, 21 references)

Yukihiko Mitsui.

Kojima, K., and Nagaya, Y. **Histochemistry of the retina.** Acta Soc. Ophth. Japan **57**:1337-1347, and 1478-1480, Nov.-Dec., 1953.

The effect of epinephrine, insulin, cortisone, ribonuclease and carbon tetrachloride on the polysaccharides, nucleic acids and phosphatases of the retina in vitro and in vivo was studied in experimental animals. In the rabbit, glycogen was caused to disappear from the liver by carbon tetrachloride, while the glycogen in the retina was little influenced. (10 figures, 4 tables, 8 references)

Yukihiko Mitsui.

Michaelson, I. C., Herz, N., and Rapoport, G. **Effect of hyaluronidase on new vessel formation in the cornea.** A.M.A. Arch. Ophth. **50**:613-617, Nov., 1953.

Hyaluronidase had no influence on new vessel growth into the cornea in controlled experiments on rabbit corneas. (9 references)

G. S. Tyner.

Yoshizawa, K. **Zymohexose in the retina.** Acta Soc. Ophth. Japan **57**:1403-1406, Nov., 1953.

Zymohexose in the retina was studied by Allen-Bourne's method (J. Exp. Biol. 20:61, 1943). The myoid of the cones and the inner segment and the ellipsoid of the rods contained a great amount of this enzyme. (1 figure, 6 references)

Yukihiko Mitsui.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Alagna, G. **The mechanism of stereoscopic vision.** Arch. di ottal. **57**:445-464, 1953.

The principal theories are reviewed and the author's own investigations are presented. According to him the ocular movements are a fundamental factor in the determination of stereoscopic vision. The nature of these movements is identified and the laws regulating their optico-geometric functions is determined. They consist of very rapid, rectilinear oscillatory movements in the direction of 135° meridian. This, according to the author, is the mechanism of stereoscopic vision: objects fixed in space produce a well-defined image on the retina. By means of the oscillating movements (preparatory phase) the contours of the objects are observed, but only in the following phase of rotation of the optic axis the evaluation of the distance becomes possible. During this second phase the eye perceives a different projection of the contours proportionate to the distance of the object. (3 figures, 38 references)

John J. Stern.

Berner, G. E., and Berner, D. E. **Relation of ocular dominance, handedness, and the controlling eye in binocular vision.** A.M.A. Arch. Ophth. **50**:603-608, Nov., 1953.

The authors believe that left or right handedness is definitely related to ocular dominance. Eye-hand confusion is likely to exist if there is a difference in the dominance of the two. (2 tables, 7 references)

G. S. Tyner.

Ellerbrock, V. J. **Magnification for near vision.** Am. J. optometry **31**:67-77, Feb., 1954.

This is a mathematical presentation of methods of magnification by means of large diameter plus lenses or mirrors before the eyes. Such devices could be used by persons with subnormal vision, or in industry where the task involves fine detailed vision. The principle of a projection magnifier to enlarge a book page five

diameters is described. Paul W. Miles.

Ginsborg, B. L. **Small voluntary movements of the eye.** Brit. J. Ophth. **37**:746-754, Dec., 1953.

A detailed investigation of the small eye movements which occur when the gaze is transferred from one fixation point to another is described Two separate processes probably occur in the movement of the visual axis towards a fixation point. The first process is concerned with establishing the visual image within the central (1°) region of the fovea. The second of two saccates is therefore comparable to the reflex response to the exposure of a new visual stimulus. Orwyn H. Ellis.

Tinker, M. A. **Light intensities preferred for reading.** Am. J. optometry **31**: 55-66, Feb., 1954.

Experiments were made on 144 university students to determine the light intensity preferred for reading. Those students who were pre-adapted to high illumination selected a high intensity for reading. Those pre-adapted to low illumination selected low intensity. The intensity preferred for reading cannot be used as a test for efficient seeing.

Paul W. Miles.

Winkelman, J. E. **Central and peripheral fusion.** A.M.A. Arch. Ophth. **50**: 179-183, Aug., 1953.

The author states that binocular vision is fusion, and this fusion should be differentiated into motor fusion (fusional movements) and sensory fusion, or the process of uniting the two retinal images into a unitary perception, as recently stressed by Burian. After discussion of his experiments, Winkelman states that in many cases of strabismus it is impossible for the retinal images to be combined into a single mental impression. In normal subjects a similar condition may be brought about by using images giving rise to retinal rivalry. When these images are observed haploscopically with the fovea of both eyes, retinal rivalry occurs. When, on the contrary, the periphery of both retinas is stimulated by retinal rivalry images, a fusional movement may occur. (16 references) R. W. Danielson.

5

DIAGNOSIS AND THERAPY

Chwirot, Roman. **Transportation of the patient after cataract extraction.** Klinika Oczna **23**:201-204, 1953.

The author stresses the danger of complications to cataract patients during their transportation from the operating room to their bed. To minimize the frequency of complications the author describes a canvas stretcher spread on a quadrangular metal frame. The canvas part is divided in two halves lengthwise and loops are placed on both edges. A wire is passed through the loops holding both halves of the canvas together. When the patient is put to bed the wire is pulled out through one end and the canvas pulled from under the patient on both sides.

Sylvan Brandon.

Cremer, Max. **The treatment with supronal in ocular diseases.** Klin. Monatsbl. f. Augenh. **124**:86-89, 1954.

The author prefers a 10-percent solution of this sulfonamide. He successfully treated a number of infectious diseases, but no details of statistics are given. (1 figure) Frederick C. Blodi.

Kawashima, K. **Pulsation curve of the cornea.** Acta Soc. Ophth. Japan **57**:1366-1369, Nov., 1953.

Kawashima analysed the pulsation curve obtained by an electric tonometer. He describes characteristic features of the curve in cases of angiospasm and

angiosclerosis of the retina. (6 figures, 7 references) Yukihiko Mitsui.

Newell, F. W. **Eye emergencies.** M. Clin. North America **38**:225-240, Jan., 1954.

Eye emergencies are covered in a very general manner for nonophthalmologists under the headings: the red eye, the painful eye, loss of vision, and ocular injuries. The importance of the recognition of glaucoma, and the general treatment of ocular injuries are well stressed.

Harry Horwich.

Nicolai, Heinz. **Further experiences with the optokinetic nystagmus as an objective method to determine visual acuity.** Klin. Monatsbl. f. Augenh. **124**:81-86, 1954.

This new apparatus had already been described (Klin. Monatsbl. f. Augenh. 122, 1953). It allows an easy, elegant and simple examination of the patient. Two cases of malingering are discussed (3 figures, 1 chart, 3 references)

Frederick C. Blodi.

Orlowski, Witold J. **Practical method of tissue therapy. Preliminary report.** Klinika Oczna **23**:175-181, 1953.

The author accepts tissue therapy as a well established method of treatment. He feels that fluid tissues are easier to handle and to administer than the solid ones. Preserved blood was his choice at the beginning but later he used only the serum, because of considerably smaller reaction than with the whole blood. It was preserved for two to seven days at 5° centigrade and one-half cc. was injected subconjuntivally every second or third day. The site of injection was chosen for psychological reasons. Five injections were given and if necessary the course was repeated in one month. Three cases of traumatic optic atrophy, one case of third degree burn and one case of injury with aniline dye were treated. Visual fields increased appreciably in all three cases of optic atrophy. Scarring in both cases of chemical injury was comparatively mild. (3 figures, 1 table, 13 references)

Sylvan Brandon.

Schillinger, Robert J. **Intravenous pentobarbital (nembutal) sedation in eye surgery.** A.M.A. Arch. Ophth. **50**:184-187, Aug., 1953.

The author describes a method of continuous drip of dilute pentobarbital (nembutal) sodium administered just before and during the operation. It is designed to give controlled sedation rather than general anesthesia. This is combined with a subcutaneous injection of 50 to 100 mg. of meperidine (Demerol) hydrochloride and routine local anesthesia. Schillinger claims satisfactory sedation in 75 of 80 operations by this method and claims that by its use nervous patients may be operated upon without resorting to general anesthesia. (5 references) R. W. Danielson.

Segal, Pawel. **New advances of hormone treatment in ophthalmology.** Klinika Oczna **23**:205-217, 1953.

The author reviews the use of cortisone and ACTH in clinical and experimental ophthalmology. (88 references)

Sylvan Brandon.

6

OCULAR MOTILITY

Calmettes, D., and Pigassou, R. **Ocular torticollis (analysis) of eight cases).** Arch. d'opht. **13**:673-678, 7, 1954.

The authors define ocular torticollis as an abnormal position of the head caused by a disturbance of ocular motility, generally a paralysis of a vertical muscle. They report eight observations in three types of cases. In the first type, a congenital torticollis with absence of fixation

of one eye, surgical intervention and visual reeducation led to disappearance of the torticollis and acquisition of stereoscopic vision in all cases. In the second type there was a congenital amblyopia with strabismus, not correctible by surgery and orthoptic treatment. In the third type there was binocular vision with the head tilted but vertical diplopia when the head was in normal position. The authors explain the cases with amblyopia and with faulty fixation on the basis of early faulty binocular vision which was later lost but which led to the torticollis. They urge early attention and report their own eight cases in detail with photographic documentation. (5 figures)

Phillips Thygeson.

Schwarz, G. A., and Liu, C. N. **Chronic progressive external ophthalmoloplegia; a clinical and neuropathologic report.** Arch. Neurol. & Psychiat. **71**:31-53, Jan., 1954.

A brief clinical report of a case of chronic progressive external ophthalmoplegia of 36 years' duration is given. A detailed pathologic study was made, with special attention given to the oculomotor, trochlear and abducens nuclei, the nerves themselves, and the superior oblique, superior rectus and lateral rectus muscles. Special sections were made and a variety of stains were used. No significant changes were found in any of these nuclei or nerves, and in the ciliary ganglion. The muscles, however, showed marked changes; there was destruction of the muscle fibers and overgrowth of fat and connective tissue. The authors concluded that chronic progressive external ophthalmoplegia is a form of muscular dystrophy. (12 figures, 35 references)

Harry Horwich.

Wilczek, Marian. **Diagnosis of paralyzed ocular muscles.** Klinika Oczna **23**: 183-188, 1953.

The author presents the basic principles of action of eye muscles. Symptoms and compensatory adjustments are described in cases of paralysis of one or more muscles. Methods of examination in paralytic squint are described. (3 figures)

Sylvan Brandon.

7

CONJUNCTIVA, CORNEA, SCLERA

Anastasi, Giovanni. **Some uses of hyaluronidase in ocular therapy.** Arch. di ottal. **57**:465-472, 1953.

In 15 patients with pterygium of various size and eight with trachomatous cicatricial pannus, a solution of 150 units of hyaluronidase was injected subconjunctivally once weekly. Small pterygia (up to 1 mm. on the cornea) were cured. Larger ones were only temporarily improved. Trachomatous pannus was temporarily cleared but no permanent improvement was obtained. (5 references)

John J. Stern.

Arkin, W., and Dryjski, J. **Treatment of corneal xerosis by transplantation of Steno's duct into the conjunctival sac.** Klinika Oczna **23**:167-170, 1953.

The authors describe transplantation of the parotid ducts into the conjunctival sacs in a patient with bilateral corneal xerosis. The result was very satisfactory, but there was very annoying watering during ingestion of food. The method used by the authors was described by Filatów.

Sylvan Brandon.

Borrie, P. **Rosacea with special reference to its ocular manifestations.** Brit. J. Derm. **65**:458-463, Dec., 1953.

The author analyzed 133 patients with cutaneous rosacea and 99 patients with combined ocular and cutaneous lesions. The disease does not spread from the eye

to the face or vice versa. Ocular lesions respond to X-ray therapy, while those of the skin do not. Therefore the lesions, although having a common cause, are independent of each other. (4 tables, 6 references) Irwin E. Gaynon.

Braley, A. E., and Alexander, R. C. **Superficial punctate keratitis.** A.M.A. Arch. Ophth. **50**:147-154, Aug., 1953.

Superficial punctate keratitis is a confusing term which has been applied to numerous superficial changes in the cornea. Fuchs first used the term to describe the corneal changes associated with epidemic keratoconjunctivitis. Since then nearly any type of change in the epithelium and superficial layers of the cornea has been classified in this category. Thygeson has published a full clinical description and differential diagnosis of 26 cases. The authors add observations about the disease. It is a bilateral keratitis characterized by epithelial and subepithelial opacities, rather centrally and superiorly located. They consist of white or gray dots and may stain with fluorescein. There is no evidence of conjunctivitis and cultures rarely yield bacteria. Any hyperemia is usually above the limbus superiorly. There are remissions. Corneal sensitivity may be decreased and lacrimation may be increased.

The method of isolation of the virus is described. The virus appears to be very small, perhaps between 50 and 60 mμ in diameter. When the killed virus is used in therapy, the results are unpredictable. No antibiotic or chemotherapeutic agent is of any value in treatment. (5 figures, 4 references) R. W. Danielson.

Breinin, Goodwin M. **Scleredema adultorum.** A.M.A. Arch. Ophth. **50**:155-162, Aug., 1953.

Scleredema adultorum is a rare systemic disease characterized by generalized nonpitting edema of the body without fever, loss of weight, or much evidence of illness. It is preceded by an acute infection, usually respiratory, and of streptococcal origin. In general reports, involvement of the lids and conjunctiva has been mentioned, but only one case has appeared in the ophthalmic literature. Another case of scleredema is here described, with chronic edema of the lids and conjunctiva for eight years. Conjunctival lymphangiectasia and trophic corneal changes were noted. The disease may be due to a change in the ground substance of the mesenchyme and must be differentiated from scleroderma and sclerema as well as from other types of edema. (4 figures, 23 references) R. W. Danielson.

Chams, Mohsenine, and Armine. **Experimental inoculation of trachoma in man with ground tarsal material.** Rev. intern. du trachome **30**:465-474, 1953.

The authors report two observations in which tarsal tissue, removed by cutaneous incision from each of two cases of active untreated trachoma, was used to inoculate the normal conjunctiva of two blind eyes. From the first inoculation a mild conjunctival inflammation, with inclusion bodies demonstrable in scrapings, developed on the ninth day. From the second inoculation a mild conjunctivitis with follicles developed on the seventh day. Inclusions were demonstrable in scrapings. The authors considered their experiments proof that trachoma virus exists in the tarsus as well as in the conjunctival epithelium. In his discussion of the paper, Bietti pointed out that it was technically difficult to exclude islands of epithelium invaginated in the tarsus. (9 figures) Phillips Thygeson.

Goldsmith, A. J. B. **The ocular manifestations of rosacea.** Brit. J. Derm. **65**:448-457, Dec., 1953.

Blepharitis, conjunctivitis and marginal ulcers, although common, are not specific. The severe nodular keratitis and conjunctivitis develop in the exposed interpalpebral region and become surrounded by persistent vascular dilatation. The incidence is most common during spring and early summer indicating that external allergens may irritate an already sensitized eye. Rosacea keratitis, once developed, progresses independently of the skin disease. (3 figures, 1 table, 29 references) Irwin E. Gaynon.

Krwawicz, T., Seidler Dymitrowska, M., and Vorbrodt, A. **Histochemical investigation of the cornea in keratoplasty.** Klinika Oczna **23**:155-166, 1953.

The authors investigated the changes occurring in the cornea of the host and in the transplant. Of particular interest was the histiocytic type of cell, which was stained according to the Weil-Davenport method. Histochemical methods were used to investigate the behavior of vitamin C, glutathione, proteins containings the SH group and ribonucleic acid. The corneal cells in the transplant sometimes show the ability of migration and even may be transformed into polyblasts. The vitality of corneal cells was demonstrated by the presence of mitosis in the transplant ten days after the transplantation. Filatow's idea that the corneal cells have histiocytic properties was confirmed. When the tissues lack proteins containing SH groups the results of transplantation were poor. Corneas removed from the donor within six hours after death and preserved in paraffin less than 24 hours contained intracellular granules of vitamin C in the epithelium. Corneas removed after six hours or preserved longer than 24 hours showed the presence of extracellular granules of vitamin C. This suggests disturbances in the vital chemical processes in the latter group of corneas. Corneas preserved according to Filatow's method showed different amounts of granules of vitamin C intracellularly depending on the length of time of preservation. (9 figures, 9 references) Sylvan Brandon.

Larmande, A. M. **Treatment of partial trichiasis and of hemi-entropion.** Rev. intern. du trachome **30**:532-535, 1953.

Each case of incomplete ciliary deviation requires analysis to determine the proper surgical technique to be applied. When only a few cilia are involved, electrolysis or diathermic coagulation can be used, whereas extensive trichiasis or distichiasis call for tarsectomy or bulbar epilation. Spontaneous hemi-entropion requires classical tersectomy. Post-operative hemientropion requires a partial tarsectomy. The author describes his modification of the operation of Trabut which has given satisfactory results. (2 figures) Phillips Thygeson.

Ljubisa, Mandic. **Clinical observations on trachoma in Podrigne.** Rev. intern. du trachome **30**:542-549, 1953.

The author describes the special characteristics of trachoma in Podrigne, Yugoslavia. It begins as a banal conjunctivitis of slow evolution. Symptoms are mild unless secondary infection occurs. Pannus is rare and blindness from the disease very unusual. Trichiasis and entropion occur only in the very old cases. Established trachoma is encountered in three forms or types, first a follicular type, second a more common papillary type, and third a mixed type. Follicles are sometimes encountered on the bulbar conjunctiva and on the semilunar fold. Limbal follicles have not been observed. The follicular form is easier to treat than the papillary and mixed forms. The author believes that antibiotics and sulfonamides act only on secondary infection and that classical methods of therapy are necessary to cure the trachoma itself. Phillips Thygeson.

Paufique, L., and Moreau, P. G. **Scleromalacia perforans. Histologic considerations and treatment with a scleral graft.** Ann. d'ocul. **186**:1065-1076, Dec., 1953.

The authors review the clinical characteristics of scleromalacia perforans. They feel that most of the cases reported in the literature are really cases of necrosing nodular scleritis. They describe a technique of scleral grafting which they used with good results in one case. Histologic examination showed that the lesion was neither an infectious process nor a rheumatic nodule, but a localized scleral degeneration. (3 figures, 7 references)

John C. Locke.

Stany-Habachi. **Anatomoclinical therapy of the trachomatous. (Therapeutique anatomo-clinique du trachomateux.)** Rev. intern. du trachome **30**:479-514, 1953.

The author reviews the history of the treatment of trachoma from the time of the ancient Egyptians to the present and concludes that no one treatment is effective in all cases. He urges an analysis of each individual case according to the extent and depth of the lesions, the amount of cicatrization, the condition of the cornea, and the presence or absence of secondary infection. He states that surface lesions are accessible to treatment with the sulfonamides and the antibiotics but that massage and expression of follicles are useful adjuncts. He believes that deep lesions require subconjunctival injections of a sulfonamide associated with hyaluronidase, together with superficial scarifications of the conjunctiva followed by energetic squeezing with the Knapp roller forceps. In old resistant cases with deep involvement he urges the use of combined tarsal-conjunctival excision but mentions that corneal complications can follow eyelid surgery. He discusses the indications and contraindications for corneal transplantation in trachoma and concludes with a consideration of the need for further therapeutic research on the disease. (57 references)

Phillips Thygeson.

Stark, Hilmar. **Cortisone and the eye.** Klin. Monatsbl. f. Augenh. **124**:96-101, 1954.

The author discusses in a very general way and without giving any details or statistics, some indications for cortone in corneal diseases. It was of advantage in clearing ulcers, metaherpetic inflammations, interstitial keratitis, and acne rosacea.

Frederick C. Blodi.

Thygeson, Phillips. **Criteria of cure in trachoma with special reference to provocative tests.** Rev. internat. du trachome **30**:450-464, 1953.

The author discusses the criteria for cure in trachoma and reports observations indicating that treatment can be safely discontinued when biomicroscopic examination reveals a disappearance of all subepithelial infiltration and epithelial keratitis, even though conjunctival changes remain. No relapses occurred in 72 cases in which treatment was stopped according to this principle.

In a small series of cases, cortisone applied topically effected activation of trachoma as evidenced by the finding of inclusion bodies. The value of this reaction as a diagnostic test of trachomatous activity warrants further exploration.

Phillips Thygeson.

Venco, L., and Citroni, M. **The survival of preserved cornea.** Ann. d'ocul. **186**: 1077-1085, Dec., 1953.

Using a polarographic method, the authors show that the oxygen consumption of corneal tissue, stored by Burki's method, decreases irreversibly after two weeks. The authors compare their results

with those of others reported in the literature. (2 figures, 3 tables, 27 references)
John C. Locke.

Wildi, G. **Epidemic keratoconjunctivitis.** Klin. Monatsbl. f. Augenh. **124**:93-95, 1954.

An acute epidemic in his practice could be interrupted by sterilization of all drops, ointments and instruments. The treatment of this disease remains unsatisfactory. Frederick C. Blodi.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Bartolome, Aguilar. **Therapy of uveitis.** Arch. Soc. oftal. hispano-am. **13**:1176-1216, Oct., 1953.

An exhaustive review of the literature is followed by an extensive bibliography.
Ray K. Daily.

Fukuda, M. **Keto-enol substances in the urine of patients with Harada's syndrome.** Acta Soc. Ophth. Japan **57**:1418-1422, Nov., 1953.

In the convalescent stage of Harada's syndrome, patients exhibit an abnormal increase in excretion of keto-enol substances in the urine. (4 figures, 10 references)
Yukihiko Mitsui.

Gonzalez-Pola, Angel Moreu. **The concept and therapy of sympathetic ophthalmia.** Arch. Soc. oftal. hispano-am. **13**: 1143-1161, Oct., 1953.

An exhaustive review of the literature on the subject confirms the author's belief that sympathetic ophthalmia is a very rare disease. He believes that the development of sympathetic ophthalmia requires the following sequence of processes. 1. External infection of the globe with exteriorization of the uvea. 2. The setting up a chronic nodular exudative uveitis, with a persistent irritability of uveal tissue. 3. Changes in the hypophyseal-hypothalamic region and in the diencephalic centers. 4. An exiciting agent of sufficient intensity to interrupt the equilibrium between the eye and the diencephalon. 5. Endocrine-neuro-vegetative changes, which affect the vascular permeability, neurocirculatory regimen, and fibroblastic cycle of both eyes. Any interference in this sequence inhibits the development of sympathetic ophthalmia; it is logical, according to the author, that a break in this sequence is common, which accounts for the rarity of the disease. Therapy should utilize pyretotherapy, topical cortisone, ACTH, sodium salicylate, gentisic acid, orbital alcohol injections, and mydriatics. Ray K. Daily.

Kimura, S. J., Hogan, M. J., and Thygeson, P. **Uveitis in children.** Am. J. Dis. Child. **87**:40-48, Jan., 1954.

In 810 cases of uveitis, 47 or 5.8 per cent, were found in patients under the age of 16. There were 29 cases of posterior uveitis, and only 18 of anterior uveitis. Despite very thorough work-up, an etiologic diagnosis was found in only six of the latter, and ten of the former, all ten presumed due to toxoplasmosis. Cases were seen of heterochromic iridocyclitis, Boeck's sarcoidosis, syphilis, Still's disease, and frank allergic diathesis. No cases were ascribed to tuberculosis. The importance of Coats' disease and heredomacular degeneration in the differential diagnosis was noted. The authors suggest that allergy may be an important factor in childhood uveitis. (8 tables, 27 references)
Harry Horwich.

Kornblueth, W., and Stein, Richard. **Case notes: sympathetic ophthalmia following an operation for retinal detachment.** Brit. J. Ophth. **37**:755-757, Dec., 1953.

The author reports the case of an 8-year-old boy with high myopia who developed almost complete retinal detachment. He was uncooperative and surgery was unsuccessful. Nine months postoperatively a severe endophthalmitis developed and one month later sympathetic ophthalmia developed in the other eye. In spite of energetic treatment including immediate enucleation of the inciting eye and cortisone the sympathizing eye became shrivelled. Orwyn H. Ellis.

Sampaolesi, R. **Fluorescein permeability test. Report on 500 observations.** Arch. oftal. Buenos Aires **28**:297-310, July, 1953.

Amsler and Huber's fluorescein test was performed in 265 normal persons and in 235 cases of diverse eye conditions. Results corresponded exactly to those summarized recently by Amsler and Huber (Ophthalmologica 121:130, 1951): permeability of the blood-aqueous barrier is neatly augmented in all traumatic and infectious conditions in which the uveal tract is involved, as well as in heterochromic cyclitis and in thrombosis of the retinal veins; no significant changes are found in chronic simple glaucoma; in cases of perforating injuries of the globe, the test proves particularly helpful in preventing the occurrence of sympathetic ophthalmia, since a more or less increased passage of the drug into the anterior chamber can be detected in the fellow eye several weeks before the outburst of any actual inflammatory phenomena. (21 figures, 18 references)

A. Urrets-Zavalía, Jr.

Santos, E. **Sympathetic ophthalmia.** Arch. chil. de oftal. **10**:38-39, Jan.-June, 1953.

The author refers briefly to the clinical history of a patient with an ocular injury which was sutured two days after the accident occurred. During one month the patient was treated with antibiotics, midriatics, and cortisone. During the entire time the patient had a violent iritis, but no signs of a cyclitis. At the end of the month the eye was enucleated. Five weeks after the enucleation, the patient returned with a severe iritis of the other eye, with posterior synechiae, but no signs of cyclitis. He was treated with cortisone, mydriatics, and antibiotics, and the eye became white in a very few days. The author reports this case to emphasize the good effects of cortisone. No pathologic examination of the enucleated eye was done. Walter Mayer.

Sourdille, G. P. **Surgical therapy of iridocyclitis.** Arch. chil. de oftal. **10**:11-17, Jan.-June, 1953.

The surgical approach to iridocyclitis is one of the most controversial problems in ophthalmology, and is to be considered only after a failure of medical treatment. One must consider the true hypertensive iridocyclitis, in which the principal factor is an elevated ocular tension, and the secondary post-iritic glaucoma, which becomes a surgical problem only after a certain time because of a pupillary block or an obstruction in the angle, as seen with the gonioscope. In other cases, it may be a severe hypotension which will lead to the surgical approach.

The most important group of cases of surgical iridocyclitis are those of anterior uveitis with a great cyclitic component, the hypertension being due to edema of the vitreous (increased water uptake) or to an angle block. The diagnosis must be made by tonometry, because visual field changes and cupping of the optic disc are late signs and difficult to evaluate in these cases. The surgical treatment will only be attempted after failure of medical treatment. The author's stand in the controversy about miotics or mydriatics in these cases is that he believes the ciliary body

should be placed at rest. Therefore, he uses mydriatics combined with cortisone, antibiotics, and local heat to relieve the local pain, a result which he also obtains by combining cocaine with the topical medications. If this therapy fails, he uses a De Weekers retrociliary diathermy operation in cases of mild hypertension, and an iridencleisis ab externo in the more severe ones, with a very slow decompression of the globe, which will also bring about a spontaneous prolapse of the iris, obviating the need of introducing instruments into the anterior chamber.

In the secondary glaucoma caused by a pupillary block due mostly to iritis, a good mydriasis will prevent the block in many cases except in the plastic forms, such as Heerfordt's disease or sympathetic ophthalmia. In these cases, a very early total iridectomy is indicated; however, further experiences with cortisone may change this opinion. The iridectomy should be ab externo, and only in very rare cases should a peripheral iridectomy be performed for cosmetic reasons.

In the last group of cases of hypertensive iritis the hypertension is due to obstruction which results from cyclitic precipitates and iritic exudates in the chamber angle. Gonioscopy is of the greatest importance here, and the author generally uses a cyclodialysis for these eyes. If one of the procedures of sclero-iridectomy is to be preferred, he uses an iridencleisis, having had bad results with Lagrange's and Elliot's procedures. In other cases, a pupillary block may lead to a diminution of ciliary secretion, and phthisis bulbi occurs if an iridectomy is not done, combined with the rest of the medical measures against uveitis. In certain cases with posterior synechiae and no changes in the tension, an iridectomy may be indicated to prevent further complications. Finally the author mentions anterior synechiae. If they are due to traumatic perforation, a lysis of these adhesions should be done. If they are due to inflammation, as in Heerfordt's disease, surgery should only be attempted after the eye is completely quiet. A cyclodialysis should be made in mild cases, or a direct section of the synechiae in the more severe forms. Walter Mayer.

Woods, Alan C. **Use of specific streptococcus vaccine in nongranulomatous uveitis.** A.M.A. Arch. Ophth. **50**:129-146, Aug., 1953.

The purpose of this paper is to report the findings and the conclusions drawn from a study of 208 patients of whom 107 had granulomatous and 101 nongranulomatous uveitis. Granulomatous uveitis is believed to be basically due to an actual infection of the tissues with the living organisms which characteristically cause granulomatous disease, while nongranulomatous uveitis is considered to be a bacterial allergic reaction. If nongranulomatous uveitis is an allergic reaction, usually due to a bacterial hypersensitivity to streptococci, one should be able to demonstrate high incidence of specific streptococcic sensitivity, greatly lessened incidence of systemic granulomatous disease in the nongranulomatous group, and control of inflammation and prevention of recurrences by desensitization of the patient. Woods found that 89 percent of patients with nongranulomatous uveitis showed a specific hypersensitivity whereas 20 percent of patients with granulomatous uveitis showed similar sensitivity. Only 10 percent of patients with nongranulomatous uveitis showed evidence of systemic granulomatous disease, and 78 percent of patients with granulomatous uveitis. The intravenous administration of a streptococcus vaccine to hypersensitive patients with nongranulomatous uveitis was therapeutically successful in approximately 80 percent of such patients. This evidence

suggests that nongranulomatous uveitis is an allergic reaction, usually dependent on allergy to streptococcus. (9 tables, 21 references) R. W. Danielson.

9

GLAUCOMA AND OCULAR TENSION

Becker, B., and Friedenwald, J. S. **Clinical aqueous outflow.** A.M.A. Arch. Ophth. **50**:557-571, Nov., 1953.

Studies were conducted to evaluate the limitations of Grant's method of tonography in patients with glaucoma. In untreated open-angle glaucoma, the findings of Grant were verified. Tonography provided a useful method of diagnosis in border-line cases. Successful filtering operations for glaucoma resulted in increased aqueous outflow. Women in the progestational phases of the menstrual cycle and pregnancy showed an increased facility of outflow. (4 figures, 22 references) G. S. Tyner.

Cascio, G., and Lodato, G. **The effect of Avagal on the blood-aqueous barrier.** Arch. di ottal. **57**:425-430, 1953.

Avagal is the Italian equivalent of Banthine. Its administration in the form of a 1 to 2-percent ointment into rabbits' eyes failed to produce any modification of the normal permeability of the blood-aqueous barrier. (2 tables, 6 references) John J. Stern.

Caselli, Francesco. **The action of Mintacol on the permeability of the blood-aqueous barrier.** Arch. di ottal. **57**:413-424, 1953.

Mintacol, a new miotic recommended for the management of glaucoma, produces a marked increase in the permeability of the blood-aqueous barrier 30 minutes after instillation into rabbit eyes. The effect remains noticeable even 72 hours after administration of the drug. It is attributed to the parasympathico-mimetic action of Mintacol which produces a vasodilation in the irido-ciliary vascular bed. The antiglaucomatous effect is due to its action on the capillaries of iris and ciliary processes which produces a hydrodynamic exchange beneficial to the hypertension of glaucomatous eyes. (4 tables, 20 references) John J. Stern.

Cristini, G., and Pagliarani, N. **Amyl nitrite test in primary glaucoma.** Brit. J. Ophth. **37**:741-745, Dec., 1953.

The fall in intraocular pressure following amyl nitrite inhalation was larger in subjects who had already spontaneously shown extensive diurnal phasic variations and in those patients in whom the action of miotics had been more pronounced. The fall was very small or absent in patients showing normal ocular tension, or during the absolute stage. In congestive glaucoma no fall in tension was observed; on the contrary, during the second phase of amyl nitrite inspiration a short rise in ocular tension above the initial values occurred. After instillation of cholinergic drugs, the fall in intraocular pressure was very small and was almost completely absent in those patients in whom ocular tension had been normalized. After blocking of stellate ganglion, the variations in tension showed greater irregularity. They were mostly characterized by a fall reaching much lower values than the basal tension, and a short rise to a higher level. It is thought that the fall in basic intraocular pressure after the inspiration of amyl nitrite is caused by a diminution of the hydrostatic capillary venous pressures in the uvea following a widening of the capillary network. The rate of decrease, moreover, may be related to the widening capacity of the uveal capillary network; in cases where no fall occurs, impossibility of amplification must be deduced. The vasodilatation elicited by these drugs,

causing both dilatation of the lumina and the opening up of new uveal vascular areas, brings about the same condition that is spontaneously present in glaucoma with normal tension. This is the reason why the fall in intraocular pressure during the amyl nitrite test performed under the action of cholinergic drugs is small or absent. Further investigations seem to be necessary. Orwyn H. Ellis.

Cristini, G., and Strazzi, A. **The control of the glaucomatous tensional variation by means of a block of the stellate ganglion.** Arch. oftal. Buenos Aires **28**:413-417, Sept., 1953.

The block of the stellate ganglion brings about a diminution of the daily tensional variations in chronic simple glaucoma. This confirms the theory that the sympathetic is the mediator for the nervous impulses which affect the small uveal vessels, and it is possible that the increased tensional variations in glaucoma are due to an alteration in the cervical sympathetic. It is possible that a neuritic process in the pericarotid plexus may precipitate an attack of increased intraocular pressure. (2 figures, 13 references) Walter Mayer.

Inostroza, Waldo. **Relative value of the ocular tension and its importance in the prognosis and therapy of glaucoma.** Arch. chil. de oftal. **10**:55-61, Jan.-June, 1953.

The author warns against considering the tension as the most important factor in glaucoma, but emphasizes also the danger of not giving enough importance to the tonometric findings, as has been done by some in the recent past. To prove his opinion, he gives the clinical history and findings on two patients, one with a unilateral chronic glaucoma, and the other with chronic simple glaucoma for which operation was done on one eye only, and shows how the tonometric readings obtained after surgery were well correlated with the other findings, such as visual field, visual acuity, and tension variations after the drinking test. The author emphasizes the usefulness of the drinking test or any similar provocative test, to control the efficiency of the surgery. (2 figures, 4 references) Walter Mayer.

Mansheim, Bernard J. **Aqueous outflow measurements by continuous tonometry in some unusual forms of glaucoma.** A.M.A. Arch. Ophth. **50**:580-587, Nov., 1953.

Tonographic studies indicate that in some forms of secondary glaucoma the increased intraocular pressure is due to increased aqueous flow. In primary open angle glaucoma the cause of increased intraocular pressure is decreased outflow. (8 figures, 8 references) G. S. Tyner.

Morate, F. H. **Uniocular medicamentous glaucoma.** Arch. Soc. oftal. hispano-am. **13**:1223-1225, Oct., 1953.

This is essentially the report of a case of acute monocular glaucoma in a woman, 26 years old, precipitated by the internal consumption of atropine for the relief of asthma. The interesting feature was the involvement of the eye which had an impeded limbal capillary circulation, and the smaller pupil. The case shows that an attack of acute glaucoma may be precipitated in an eye susceptible to the disease by the internal administration of atropine. For this reason the author urges that atropine be sold only on prescription. Ray K. Daily.

Posner, Marvin. **Transient glaucoma associated with meningeal hydrops.** A.M.A. Arch. Ophth. **50**:189-191, Aug., 1953.

The author reports in detail a case of glaucoma which occurred during the acute phase of meningeal hydrops. The

nature of the episode is not clear, but it seems likely that the glaucoma was somehow associated with and related to the systemic disease. An interesting feature was the change in the discs, from a borderline normal condition, at the time of elevated ocular tension, found on the first examination, to full-blown papilledema, a few days after the ocular tension became normal. The author wonders whether the increased tension was sufficient to suppress the papilledema.

R. W. Danielson.

Thiel, R. **Etiology of primary glaucoma.** Arch. chil. de oftal. **10**:1-10, Jan.-June, 1953.

The author warns against considering glaucoma as due only to certain local factors, such as shallowness of the anterior chamber, and emphasizes the necessity of considering the eye as an organ of vegetative nervous regulation. Therefore, it must be considered in correlation with the entire organism, and the disturbed ocular tension must be considered the result of local, endocrine, and nervous factors. Each vegetative function in the body is regulated by the principle of the "double security," which means that there are two types of stimuli from the center, hormonal and reflex nervous stimuli. The author then reviews the four fundamental biological laws regarding the relations between periphery and vegetative centers.

He analyzes the action of certain substances such as Gynergen, Dolantin, and Evipan on the ocular tension and believes that their hypotensive action in acute and chronic glaucoma is due to their hypnotic action. To prove this point, the author has used certain substances, such as Pervitin, which are antagonistic to the previously mentioned hypnotics, and he was able to obtain an increase in the ocular tension after a subcutaneous injection of this substance. The third group which he analyzed were substances such as Pendiomid, which block the transmission of the stimulus in the peripheral autonomous ganglions; he found that the retrobular injection of this substance was able to reduce the ocular tension which was abnormally increased in the various forms of glaucoma.

The author discusses the factors which are responsible for the peripheral regulation of the ocular tension. He emphasizes the fact, which has not received the attention it deserves, that when the ciliary muscle contracts, the choroid also contracts. This will influence the content of the choroidal vessels, which is also a factor in glaucoma. He then emphasizes the importance of the sensitive preso-receptors, which prevent sudden changes in the ocular tension. He speculates that there must exist a system of two antagonistic hormones, unidentified as yet, which regulate the intraocular pressure.

The author analyzes the circulation of the aqueous and its relation to the intraocular pressure. The next factor analyzed is the vitreous and its function in the eye, emphasizing its formation by the fine filaments which cross each other, and by another system, recently discovered, of polygonal gel fibers. If these fibers are destroyed, the viscosity and capability of water retention are lost.

Analyzing a large series of glaucomatous patients, the author found that primary glaucoma occurs more frequently in older persons, and the hypermetropes of this group had glaucoma ten years earlier than the emmetropes or myopes. He believes that the profound transformations of the ciliary body after 35 years of age are greatly responsible for the appearance of glaucoma. Also, in such senile eyes, the vitreous gel is altered and its polygonal fibers destroyed; therefore, the vitreous will have an unlimited capability of water uptake, which explains the fact that de-

hydration reduces the tension in the glaucomatous eye. Walter Mayer.

Tyner, G. S., and Scheie, H. G. **Mechanism of the miotic-resistant pupil with increased intraocular pressure.** A.M.A. Arch. Ophth. **50**:572-579, Nov., 1953.

Clinical and laboratory experiments were undertaken to show the site of disturbance responsible for the dilated pupil seen in primary acute narrow-angle glaucoma. By a process of elimination, the authors believe that the pupil dilates during attacks of acute narrow-angle glaucoma because of some interference with sphincter muscle action at the level of the muscle cell itself. (2 figures, 7 tables, 4 references) G. S. Tyner.

10

CRYSTALLINE LENS

Arruga, Alfredo. **Dislocation of Soemmering's ring.** Arch. Soc. oftal. hispanoam. **13**:1162-1169, Oct., 1953.

This is a comprehensive review of the literature on dislocated disc-shaped cataract and a tabulated report of 12 cases collected from the literature, and one observed by the author. In eight of these the cataract was dislocated into the anterior chamber. In the author's case the cataract developed after a perforating injury and was dislocated into the anterior chamber; there were no symptoms attributable to the dislocation; the eye was hypotensive, probably myopic, the fundus was invisible, and the cataract was dense, white and probably calcified. (7 figures) Ray K. Daily.

Cogan, D. G., Donaldson, D. D., Goff, J. L., and Graves, E. **Experimental radiation cataract.** A.M.A. Arch. Ophth. **50**: 597-602, Nov., 1953.

This is a report of the effects on the lenses of rabbits' eyes of a single exposure to X-rays and neutrons at lower dose levels. Abnormalities were noted after 75r of X-radiation and 1.0×10^7 neutrons/cm^2. The authors conclude that the changes in the lens from radiation do not have a critical threshold. Lens opacities do occur from these low dosages, but they do not necessarily interfere with vision and cannot therefore be termed cataractogenic doses. A true cataractogenic dose has been arbitrarily set at 500r of X-radiation of 1.2 mev. energy and approximately 8.4×10^9 for neutrons of 14 mev. energy and 3.2×10^{10} for neutrons of degraded fission energy. (5 references) G. S. Tyner.

De Berardinis, E. **Clinical observation and experimental investigations concerning extracts of fish lenses.** Arch. di ottal. **57**:437-444, 1953.

Twenty-eight patients with cataract in various stages of maturity were treated with extracts of fish lenses. The results were entirely negative. (2 figures, 1 table, 15 references) John J. Stern.

Dunnington, J. H., and Regan, E. F. **Absorbable sutures in cataract surgery.** A.M.A. Arch. Ophth. **50**:545-556, Nov., 1953.

The authors present evidence in favor of the use of mild chromic gut in cataract surgery. It may be more desirable than silk because of the lack of focal necrosis. The suture remains intact for at least seven days. The recommended suture is 6-0 mild chromic as two border-to-border sutures. It should be placed in the superficial half of the sclera and cornea. (6 figures, 26 references) G. S. Tyner.

Ham, William T. **Radiation cataract.** A.M.A. Arch. Ophth. **50**:618-643, Nov., 1953.

This review deals with the effects of radiation in the formation of cataract in

human subjects exposed to radiant energy in their occupation and as a result of atom bomb explosions. Experimental studies in animals are also reported. Much of the information is derived from the proceedings of the 1st, 2nd, 3rd, and 4th Conferences on Radiation Cataracts under the auspices of the National Research council. (76 references)

G. S. Tyner.

Hertzberg, R. **Disseminated neurodermatitis and cataract.** M. J. Australia **1**: 36-38, 9, Jan., 1954.

The author defines atopy and discusses its relationships to neurodermatitis disseminata, of which cataract is a complication in approximately 10 percent of cases. A case report is included describing the successful extraction of one cataract. Such cataract surgery is said to be frequently followed by unfavorable complications.

Ronald Lowe.

Moses, C., Linn, J. G., Jr., and Allen, A. J. **Experimental production of radiation cataracts by fast neutrons.** A.M.A. Arch. Ophth. **50**:609-612, Nov., 1953.

Studies were conducted on dogs and rabbits to determine the fast-neutron dose after a single exposure which would result in the development of cataracts; and whether a small neutron dose repeated at frequent intervals had a greater or less tendency to produce cataracts than a single large dose. Although no definite conclusions were reached, 6 to 8 mev. neutrons of 60 to 150 N did not produce cataracts in a 2-year observation period. 810 to 900 N resulted in cataract formation in 60 to 75 percent of adult dogs. (2 tables, 3 references) G. S. Tyner.

Nutt, A. B. **Lens extraction in diabetic patients.** Brit. J. Ophth. **37**:725-730, Dec., 1953.

Wound healing is not a problem with the use of careful asepsis, antibiotics and chemotherapy when necessary, the routine administration of vitamin C and careful suturing of the wound. The last favors rapid repair and it helps to prevent postoperative bleeding. Diabetic patients are more likely to bleed than normal persons of the same age. The author describes the observation of the capillaries at the base of the finger nails. Contractility to the stimulus of trauma can be demonstrated in a normal individual by touching a capillary loop with a fine needle; this causes the capillary to close down rapidly, but this does not occur if the capillaries are abnormal, as they may be in the diabetic patient. This accounts for the fact that in a diabetic patient in whom capillary contractility is shown to be abnormal, hemorrhage is likely to persist. In the promotion of wound healing the use of ascorbic acid is valuable in normal patients, and more especially so in diabetics. Provided the diet is suitable there seems to be no factor in diabetes to hinder normal absorption of vitamin K from the small intestine. It is worth noting that such preparations as rutin and vitamin E appear to produce no sustained definite improvement in capillary fragility. To avoid iridocyclitis it is best to remove the lens in its capsule.

Orwyn H. Ellis.

Sapuppo, Concetto. **The effect of fish lens extracts on experimental naphthaline cataract.** Arch. di ottal. **57**:481-487, 1953.

The administration of fish lens extracts failed to influence the development of naphthaline cataracts in rabbits. (1 table, 17 references) John J. Stern.

11

RETINA AND VITREOUS

Bellavia, M., and Lodato, G. **The treatment of some chorioretinal diseases with**

organ-specific lipoid extracts. Arch. di ottal. **57**:431-435, 1953.

Retinal lipoid extracts were used in the treatment of 20 cases of chorioretinal atrophy in high myopia and in 11 cases of chorioretinal dystrophy of various pathogenesis. (One subcutaneous injection daily for 10 to 20 consecutive or alternating days.) In most cases a favorable result was obtained with an average visual improvement of one to two lines of the Snellen chart. The results are due to a restitution of the primitive structure of the cellular protoplasm with partial or total anatomical and functional repair. (12 references) John J. Stern.

Bougas, A. **Two cases of pseudorecurrence of retinoblastoma.** Ann. d'ocul. **186**: 1086-1092, Dec., 1953.

If a limited orbital mass occurs in a child within a few months of enucleation for retinoblastoma, orbital exenteration should not be decided upon before confirming the diagnosis of malignancy by biopsy. Two cases support this point of view. The apparent new growths were found in reality to be benign cysts arising from modified glandular elements. (2 figures, 15 references) John C. Locke.

Drozdowska, Stanislawa. **Disciform degeneration of the macula (Junius-Kuhnt).** Klinika Oczna **23**:195-200, 1953.

The author presents two cases of disciform degeneration of the macula of the Junius-Kuhnt type, in two women, 28 and 31 years of age. In both cases hemorrhages were present in the macula. In one of them the second eye became involved a year later. It was found that the nonprotein nitrogen was slightly increased and the bleeding and clotting time was longer than normal. The treatment consisted of blood transfusions and diet. Hemorrhages and exudates were absorbed quickly but the central vision was permanently destroyed. (2 figures, 3 references) Sylvan Brandon.

Roveda, Jose M. **Bilateral congenital cysts of the vitreous.** Arch. oftal Buenos Aires **28**:399-407, Sept., 1953.

After emphasizing the extreme rarity of this disease, the author tabulates in a chronological order all of the 43 case reports which so far have appeared in the literature. He then describes his case, in which there was a small cystic mass situated near the optic disc in each eye, larger in the left, and adherent with strands to the underlying retina. In the right eye, there were also some visible strands into the vitreous. Both of these cysts were movable with the motions of the globe, but always returned to their primary position. In fundus biomicroscopy, the cysts very much resemble the hyaloid apparatus. The author believes that his case represents a cystic transformation of hyaloid remnants. He emphasizes the frequent association of such structures with retinitis pigmentosa. (4 figures, 43 references) Walter Mayer.

Sapuppo, Concetto. **Diascleral diathermo-coagulation of the choroid in Coats' disease.** Arch. di ottal. **57**:473-479, 1953.

A 20-year-old patient with Coats' disease and retinal detachment was treated with diathermic punctures and surface coagulations. Vision improved from 1/60 to 1/30 and the field study showed a disappearance of a central scotoma. The improvement was maintained during three months' observation. (2 figures, 7 references) John J. Stern.

Verdaguer, J., Cumsille, E., and Olivares, M. L. **Diabetic retinopathy.** Arch. chil. de oftal, **10**:18-27, Jan.-June, 1953.

In the first part of this report the clinical aspect of the disease is discussed, and

in the second part, the pathogenesis. As long as the diabetes is uncomplicated by hypertension or renal disease, the diabetic retinopathy has a pathognomonic picture. The first changes are the micronaneurysms, which the authors discuss at length, emphasizing the fact that they may be present in other diseases besides diabetes. They then discuss the diabetic exudates and the changes in the arteries and veins.

In discussing the complicated pathogenesis of the diabetic retinopathy, the authors analyze all established theories. They point out that it seems that the length of time the diabetes exists has more importance in the development of retinopathy than the actual values of glucose in blood. They deny that the renal complications or the hypertension are mainly responsible for the characteristic lesions, because many diabetics have neither of these aggravating factors. The authors refer to their experiments with pancreatectomized dogs which survived with insulin. After 25 days of survival, there was a severe lipid degeneration of the liver and kidneys but no retinopathy. There will be a later report on such dogs kept alive for eight months, which is the minimum time required to obtain a retinopathy. They speculate that if the ingestion of lipids is kept at a minimum compatible with life, it might be possible to prevent the appearance of the diabetic retinopathy. (8 references)

Walter Mayer.

12

OPTIC NERVE AND CHIASM

Arkin, W., Ficka, M., and Hryniewska, H. **Treatment of retrobulbar optic neuritis with mild hypoglycemic shocks.** Klinika Oczna **23**:171-174, 1953.

Mild hypoglycemic shocks were used by the authors in 12 cases of optic neuritis, mostly of the retrobulbar type. The fasting patient was given 30 to 40 units of insulin. In about 20 to 30 minutes symptoms of a mild shock, such as drowsiness, perspiration, restlessness and weakness, would appear. A glass of tea containing six to eight teaspoonfuls of sugar would restore the patient to normal. Five to seven shocks given every second day were necessary for recovery. Six cases where etiology was unknown had complete recovery. In the four remaining, with known etiology, the results were negative. The authors feel that the cases successfully treated were early forms of multiple sclerosis and that the shock treatment improved the circulation. Sylvan Brandon.

Asayama, R., Kashiwai, T., Shirakami, T., and Otani, M. **A combination of pituitary anterior hormone with vitamin B_1 in the treatment of glaucomatous optic nerve atrophy.** Acta Soc. Ophth. Japan **57**:1370-1377, Nov., 1953.

After this treatment the vision in glaucomatous optic nerve atrophy can improve. Repeated intraarterial injection into the carotid is most effective. (1 table, 9 references) Yukihiko Mitsui.

Espildora-Luque, C. **General considerations regarding optic neuritis.** Arch. chil. de oftal. **10**:28-37, Jan.-June, 1953.

In a series of twenty-two patients with optic neuritis all had a severe diminution of visual acuity in one or both eyes, which was sometimes recovered completely. Some had a permanent loss and some died. Sometimes headaches may precede the onset of neuritis for about two months, or may be associated with it. There will be some changes in the disc and the recovery of normal visual acuity is much more doubtful than in the cases without headaches. In many of the severe cases which resisted treatment, drainage of the sinuses brought about a complete re-

covery. In another group the author thinks that a virus played a role in the development of the neuritis because all recovered with the administration of aureomycin and vitamin B_1. He also reports two cases of neuritis produced by cerebral cysticercosis. Finally the author discusses a typical picture of optic neuritis produced by a brain tumor, and emphasizes the necessity of not overlooking this possibility in each case of optic neuritis, especially in the ones associated with headaches. Walter Mayer.

13

NEURO-OPHTHALMOLOGY

Lange, F. **The change of the pupillary reactions with age.** Klin. Monatsbl. f. Augenh. **124**:76-81, 1954.

The author measured the amount of pupillary dilatation in 196 eyes treated with mydriatics before a cataract extraction. The pupil can be maximally dilated even in the oldest patient and this speaks against a mechanical cause (e.g. hyalinization of the iris muscles) of the senile miosis. However, more and stronger mydriatics are necessary to dilate the pupil of an old patient. The author assumes a decreased tonus of the vegetative nervous system. (1 chart, 5 references)
Frederick C. Blodi.

Murray, R. G., and Walsh, F. B. **Ocular abnormalities in poliomyelitis and their pathogenesis.** Canad. M.A.J. **70**:141-147, Feb., 1954.

Choked discs occur in the convalescent stage more commonly than is believed. The explanation is not known but is in some way dependent upon the increased spinal fluid protein and the marked perivascular lymphocytic infiltration. Optic neuritis probably does not occur in this disease. Visual depression is associated with extensive brain stem damage. Visual acuity returns with survival of the patient. The most common ocular complication is horizontal nystagmus, recovery from which is the rule. Any muscle palsy may be present; it is usually the lateral rectus that is paralyzed, less frequently there is a partial third nerve palsy due to a supranuclear lesion, and rarely the superior oblique muscle is inactive. The eyelids may be affected. Opsoclonia has been observed and may be an indication of early acute anterior poliomyelitis. Any pupillary abnormality may occur except dilated fixed pupils. (1 table, 22 references)
Irwin E. Gaynon.

Nielsen, J. M. **Ophthalmoplegia externa.** Bull. Los Angeles Neurol. Soc. **18**:197-199, Dec., 1953.

Ophthalmoplegia externa is usually caused by myasthenia gravis, multiple sclerosis, cavernous sinus thrombosis, progressive muscular atrophy, muscular dystrophy, syphilis or encephalitis. Two cases of chronic ophthalmoplegia externa occurred in patients who at one time gave a positive reaction to the prostigmine test, but the response was not maintained under therapy. A patient without myasthenia gravis may give a temporary test response and lead one astray in diagnosis.
Irwin E. Gaynon.

Payne, F. **Neuro-ophthalmology.** A.M.A. Arch. Ophth. **50**:644, 666, Nov., 1953. The related literature is reviewed. (65 references) G. S. Tyner.

Prskavec, F. **Symptoms and therapy of two newer eye diseases.** Klin. Monatsbl. f. Augenh. **124**:49-58, 1954.

The first entity, called "vegetative dystonia," is a pathologic increase in the tonus of the ciliary muscle. The patients complain of visual disturbances and asthenopia. The overaccommodation is tested with the cobalt-light of Roessler. The

local treatment with digitalis is advocated.

A nasociliary neuritis is the second condition described. It causes pain in and around the eye with photophobia and lacrimation. The treatment consists of local physical therapy together with the percutaneous and subcutaneous application of snake venoms. (7 graphs, 12 references) Frederick C. Blodi.

Santos, E. **Aureomycin in neuritic diseases.** Arch. chil. de oftal. **10**:40-41, Jan.-June, 1953.

The author gives the clinical history and findings on two patients with retrobulbar neuritis and one patient with a paresis of the left sixth cranial nerve. These disorders were thought to be caused by a virus. No other etiology could be found. The patients were given treatment with aureomycin for a few days; all recovered completely in a very short time. Walter Mayer.

Sprofkin, Bertram E. **Adie's syndrome and the Adler-Scheie mecholyl test.** Neurology **3**:910-915, Dec., 1953.

Three cases of Adie's syndrome are presented, one accompanied by psychoneurosis, one by amyotrophic lateral sclerosis, and the third by late latent lues. A review of the literature indicates that the second case is a unique association. The mecholyl test was considered to be quite reliable, and very useful in the differential diagnosis of Adie's pupil. (2 figures, 21 referencs) Harry Horwich.

Wagener, Henry P. **Pseudotumor cerebri.** Am. J. M. Sc. **227**:214-225, Feb., 1954.

The author favors the term "intracranial hypertension of unknown cause" for the symptom complex characterized by papilledema and other signs of increased cerebrospinal fluid pressure in previously healthy persons without known cause. In this syndrome there are no signs of focal damage to the brain, and the mental state is bright and clear except when the pressure is very high. The spinal fluid is normal, except for the pressure; the ventricles are small and symmetrically placed; a bulging may appear after subtemporal decompression, but disappears in a few weeks although it may recur. The prognosis is good except for possible optic atrophy, and no demonstrable cause is found even at necropsy. The author mentions a recurrent case controlled by antihistaminics, and thinks the condition is one of cerebral edema which may be due to various causes. Three-fourths of the reported cases occur in women, and papilledema can occur at the onset of menstruation. Otitic hydrocephalus—a complication of otitis media—and thrombosis of the lateral sinus and the superior longitudinal or sagittal sinus are other separate conditions. Treatment consists of restriction of fluid intake and dehydration, and spinal punctures which are repeated when necessary. If this is unsatisfactory or an optic atrophy appears, a subtemporal decompression should be done. (42 references) Harry Horwich.

14

EYEBALL, ORBIT, SINUSES

Hartmann, Karl. **Symmetrical pseudotumors of the orbits in myelogenous leukemia.** Klin. Monatsbl. f. Augenh. **124**: 38-45, 1954.

Bilateral exophthalmus developed in a six-year-old girl with acute myelogenous leukemia. The cornea of the left eye developed an exposure keratitis. Irradiation of the orbits reduced the exophthalmus for the remaining two months of life. (2 figures, 16 references)

Frederic C. Blodi.

Nover, A., and Goebel, A. **Bilateral exophthalmus in rare systemic diseases.**

Klin. Monatsbl. f. Augenh. **123**:29-38, 1954.

Three cases are reported. A 57-year-old man had a bilateral exophthalmos with Hand-Schuller-Christian disease. The patient died without developing defects in the bony skull. The other two patients, a 40-year old woman and a 60-year old man, were diagnosed as giant follicle lymphoma after biopsy of the orbital mass. (5 figures, 1 chart, 34 references)

Frederick C. Blodi.

Ocampo, A. O., Grammatico, A. D., Galante, E., and Taullard, J. A. **Cholesteatoma of the orbit.** Arch. oftal. Buenos Aires **28**:289-296, July, 1953.

Primary orbital cholesteatoma is a rare cystic tumor, which develops subperiostally in relation to the roof of the orbit and produces a typical osseous defect which may be demonstrated radiologically. Surgical removal may be required if pressure symptoms or proptosis are serious enough to endanger vision or to preclude binocular vision.

The case of a 62-year-old man is presented, who suffered from a mild diabetes and exhibited a painless, slowly growing, tumor mass in the upper and outer part of the orbit. The eyeball was displaced forward and downwards; the exophthalmos could not be reduced by digital pressure; convergence was seriously handicapped and elevation abolished. Fundus examination showed a slight papilledema, as well as some retinal folds due to scleral indentation. Vision was unimpaired. Physical examination was negative, and only a slight hypercholesterolemia was recorded.

The tumor was successfully removed through a marginal orbitotomy, and subsequent pathological examination confirmed the pre-operative, tentative diagnosis of orbital cholesteatoma. (7 figures, 8 references) A. Urrets-Zavalía, Jr.

Psenner, L. **Additional remarks on the X-ray diagnosis of the skull.** Klin. Monatsbl. f. Augenh. **124**:89-93, 1954.

The author elaborates on the paper of Gigglberger (Klin. Monatsbl. f. Augenh. 121:385, 1952). The linea innominata is not the densest part of the major wing of the sphenoid, but a tangential projection of a part of the lateral orbital wall. The oblique view of the orbit (Rhese) is important for an evaluation of the apex of the orbit and of the optic canal. (3 figures)

Ferderick C. Blodi.

Rundle, F. F., Finlay-Jones, L. R., and Noad, K. B. **Malignant exophthalmos; a quantitative analysis of the orbital tissues.** Australasian Ann. Med. **11**:128-135, Nov., 1953.

The orbital tissues from a patient with malignant exophthalmos were studied histologically and biochemically, and compared with those in normal and thyrotoxic subjects. In malignant exophthalmos there was a pronounced absolute increase in the bulk of the orbital tissues which depended wholly on enlargement of the extrinsic eye muscles. Individual muscles were increased two to five times their normal weight. Histologically the muscle fibers were not increased in number but were swollen and degenerate; these changes were associated with much increase in the connective tissue elements. No increase in water content indicative of muscle edema was found.

The changes in the orbital tissues in malignant exophthalmos differ considerably from those in thyrotoxicosis with or without eye signs. In classical Graves' disease the exophthalmos is mainly produced by a deposition of fat affecting the adipose tissue between the muscles. Enlargement of the muscles makes a comparitively small contribution to the total bulk increase. Further, the exophthalmos produced in guinea pigs by the injection of

thyrotropic hormone differs from both the mechanisms described above as it is dependent on edema of the orbital tissues.

Ronald Lowe.

15

EYELIDS, LACRIMAL APPARATUS

Farina, R. **Plastic repair of superciliary alopecia.** Arch. oftal. Buenos Aires **28**: 326-330, July, 1953.

A most evident cosmetic blemish, absence of the eyebrows, can be repaired more or less successfully, if unilateral, by rotating a hair-bearing pedicle taken from the intact brow. In bilateral cases, a free graft from the retro-auricular part of the scalp may be used; the graft has to be placed into the recipient bed in such a way that the hair will grow in the proper direction. (14 references)

A. Urrets-Zavalia, Jr.

Hughes, Wendell L. **Aspiration of the lacrimal sac in acute dacryocystitis.** A.M.A. Arch. Ophth. **50**:188, Aug., 1953.

Hughes refers to a report by Cusick where penicillin was injected into the lacrimal sac by means of a hypodermic needle. Hughes describes his method of washing out the pus and instilling an antibiotic by means of a cannula through the punctum and canaliculus. He claims that one treatment will often control the acute attack, so that the obstruction of the nasolacrimal duct may be taken care of by a dacryocystorhinostomy. (1 reference)

R. W. Danielson.

Lama San Martin, G. **Acute non-suppurative recurrent dacryoadenitis ascribed to infection in an impacted tooth.** Arch. chil. de oftal. **10**:42-43, Jan.-June, 1953.

The author reports the clinical history and findings in a case of acute dacryoadenitis, which cleared completely in a few days with local application of heat, and antibiotic treatment, but afterwards recurred twice within a short period before an impacted tooth, found by X-ray examination to be infected, was extracted. There was no further recurrence after the extraction. (1 figure)

Walter Mayer.

Leahey, Brendan D. **Simplified ptosis surgery.** A.M.A. Arch. Ophth. **50**:588-596, Nov., 1953.

The author describes a method of correcting ptosis due to complete levator paralysis with normal frontalis action. The lid is suspended to the frontalis with a single 2-0 white silk suture. After resection and advancement of the levator, the use of three temporary stay sutures from the lid margin to the eyebrow is valuable in obtaining an adequately wide palpebral fissure. (9 figures, 12 references)

G. S. Tyner.

Macomber, W. B., Wang, M. K., and Gottlieb, E. **Epithelial tumors of the eyelids.** Surg. Gynec. & Obst. **98**:331-342, March, 1954.

Sixty cases of epithelioma of the lid are reviewed. The incidence was greatest in the sixth decade, and slightly higher in males. Sixty percent involved the lower lid, and twenty percent the inner canthus. Seventy percent were basal cell epitheliomas, nine of which involved deep structures. The authors preferred surgical treatment for various reasons which they list. The functional anatomy is briefly reviewed, and then the types of procedure summarized. These were excision and closure, excision and full thickness skin grafting, excision and flap shift, radical excision with enucleation of the orbit, excision and subtotal and total eyelid reconstruction, and finally the author's own technique of excision and total and subtotal reconstruction using stainless steel wire. This latter is a modification of the Hughes technique, is done in three stages, and may or may not be done with

eyelash transplantation. (16 figures, 16 references) Harry Horwich.

Taub, Eugene F. **Diagnosis and treatment of carcinoma of the eyelids.** J.A.M.A. **154**:9-12, Jan. 2, 1954.

In a discussion of carcinoma of the eyelids, 33 patients with 50 lesions, 36 of which were on the eyelids, are considered. Ten of the patients had been previously treated by other techniques. The author advocates scalpel biopsy under local anesthesia, followed by thorough curettement of the base and margins of the lesion, taking care to clean out all the finger-like pockets. Then he irradiates the open wound immediately after removal of the diseased tissue, usually giving 1,500 to 2,500 r of low voltage (100 Kv) unfiltered X-rays, or equivalent radium emanation. This achieves better than 95-percent cure. He considers ocular complications of this radiation to be unimpressive, except for the possibility of radiation cataract. He advises shielding to prevent this. (1 table, 17 references) Harry Horwich.

Truchot, P., Offret, G., and Chauvet, P. **Radiography of the normal and pathologic nasolacrimal canal.** Arch. d'opht. **13**:679-689, 1954.

The authors employ a technique of radiography of the nasolacrimal canal develeoped by Brunetti, by Kopylow, and by Toth in which an ordinary dental film is placed in the mouth between the teeth against the check and perpendicular to the rays which come from above. An exposure of 110 to 120 milliseconds with 60 kW and 20 mA is used. Interpretation of the films appears to be difficult but is described in detail with numerous photographs. In a good film only the bony contour of the lower orifice of the canal is sharp because it is close to the film. In the majority of cases it has an anterior-posterior diameter of 7 to 10 mm. and a transverse diameter of 4 to 6 mm. The superior orifice also has an oval form but of a lesser diameter (A. P. diameter 4 to 5 mm., transverse diameter 2 to 4.5 mm.). It is more difficult to measure. In a case of chronic dacryocystitis, films revealed a canal of very small diameter which no doubt favored the development of the stenosis. The films were found to be useful in revealing the lesions of periostitis secondary to syphilis, atrophic rhinitis, or repeated probing. The authors conclude that the method merits a place of choice among the various techniques available for exploration of the nasolacrimal passages. (18 figures, 18 references) Phillips Thygeson.

16

TUMORS

Krohmer, J. S., Thomas, C. I., Storaasli, J. P., and Friedell, H. L. **Detection of intraocular tumors with the use of radioactive phosphorus.** Radiology **61**:916-921, Dec., 1953.

Thirty-two ocular lesions were studied, using a Geiger counter with a special small end-window. Counts were made immediately after intravenous injection of 500 microcuries of P^{32}, and then repeated several times. The counter was applied directly over the lesion when possible, and then over the opposite pole. After enucleation, histologic sections and radioautographs were prepared, and samples of the tumor, sclera, retina and lens were assayed for radio-activity. The authors concluded that a selective uptake ratio of over 1.4 in the anterior segment was very suggestive of tumor, and less than 1.2 was strongly against it. Posterior lesions were not as definite, since 5 mm. or more between the tumor and counter was found to be very unsatisfactory. (2 figures, 1 table, 7 references) Harry Horwich.

Lagos, Eduardo J. J. **Tumors of the**

limbus and ocular adnexa. Arch. oftal. Buenos Aires **28**:385-398, Sept., 1953.

The author published an essay on this same subject in 1936, and mentions all tumors of the limbus or of the adnexa he has seen since. He also summarizes the findings and opinions of many other authors. He recommends that a biopsy be done. If the results show that the tumor is of a malignant type, early extirpation is advised. The area is then treated with radium therapy, especially if the pathologist's report shows that it was an epithelioma. If the tumor is situated at the limbus, the radium application should be made in a tangential way, in order to prevent injury to the lens. If necessary, the patient may be given another series of radium applications three months later. (5 figures) Walter Mayer.

Mann, I., Yates, P. C., and Ainslie, J. P. **Case notes: unusal case of double primary orbital tumor.** Brit. J. Ophth. **37**:758-762, Dec., 1953.

Surgical removal of an astrocytoma of the left optic nerve in a child was followed by deep X-ray therapy. The globe was left in situ, but the treatment was followed by a deep X-ray burn. Six years later the diagnosis of meningioma arising from the dura over the left frontal lobe was made. Although considerable destruction of the frontal bone was present, the tumor was apparently removed completely. Nine months later the meningioma was found to have recurred so extensively that removal was not possible even though an exenteration was done. Six months after the last operation (15 months after the primary meningioma was found) the patient died. The literature on multiple primary tumors, which are very rare, is reviewed.

Orwyn H. Ellis.

Müller, Horst. **Diffuse choroidal tumor with the clinical picture of a focal chorioretinitis.** Klin. Monatsbl. f. Augenh. **124**: 45-49, 1954.

A 53-year-old man complained of loss of vision in one eye. The ophthalmoscopic picture was interpreted as chorioretinitis, but when the eye was enucleated two years later, a flat, diffuse tumor of the choroid was found. This was diagnosed as a neuro-ectodermal tumor. (3 figures, 8 references) Frederick C. Blodi.

17

INJURIES

Glees, M. **Ocular injuries of boxers.** Klin. Monatsbl. f. Augenh. **124**:101-103, 1954.

This is a short review of pugilistic injuries to the eye. Practically any part of the eye can be injured and the protection by the superciliary arc is only a relative one. (3 references)

Frederick C. Blodi.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month. To receive adequate publicity, however, notices of postgraduate courses and meetings should be received at least three months before the date of occurrence.

DEATHS

Dr. Charles Edward Bauer, Poughkeepsie, New York, died December 9, 1953, aged 61 years.

Dr. Vernon Alaska Chapman, Palm Springs, California, died January 15, 1954, aged 80 years.

Dr. George Lester Kilgore, San Diego, California, died January 13, 1954, aged 51 years.

Dr. Avery De Hart Prangen, Rochester, Minnesota, died January 30, 1954, aged 63 years.

ANNOUNCEMENTS

ORTHOPTIC EXAMINATIONS

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in July and September, 1954.

The written examination will be nonassembled and will take place on Thursday, July 22nd, in certain assigned cities and offices, and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, September 18th in New York City, just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Applications for examination will be received by the office of the secretary of the American Orthoptic Council, Dr. Frank D. Costenbader, 1605 22nd Street, N.W., Washington 8, D.C., and must be accompanied by the examination fee of $30.00. Applications will not be accepted after June 1, 1954.

EYES NEEDED

The Uveitis Laboratory, Medical Research, Department of Ophthalmology, University of California Medical Center, San Francisco 22, California, urgently requests all physicians to send freshly enucleated eyes from cases of diffuse uveitis, iridocyclitis, and chorioretinitis for cultural and pathologic studies. The eyes should be placed in a sterile bottle, *not* fixed in formalin, and sent as quickly as possible, air express collect, to Dr. Michael J. Hogan, director, Francis I. Proctor Foundation for Research in Ophthalmology. A complete report will be sent on each specimen received.

They would also like to obtain formalin-fixed specimens of similar eyes for pathologic study.

DEDICATION PROCTOR LABORATORIES

The Department of Ophthalmology of the University of California School of Medicine at San Francisco announces the dedication on September 29, September 30, and October 1, 1954, of the new laboratories of the Francis I. Proctor Foundation for Research in Ophthalmology which was established in 1946 by Mrs. Proctor in memory of her husband.

These laboratories, whose construction was made possible through the generosity of Mr. and Mrs. Berthold Guggenhime, occupy an area of 3,000 square feet in the new Medical Sciences Building which is nearing completion. They will provide facilities of ophthalmologic research in microbiology, pathology, physiologic chemistry, and physiologic optics. Other research activities of the Foundation will be conducted in the E. S. Heller Laboratories which were donated in 1941 for research in ophthalmology and have been temporarily occupied by the Proctor Foundation pending the completion of the new building.

On Wednesday afternoon, September 29th, the staff of the Department of Ophthalmology and of the Proctor Foundation will hold open house at the new laboratories, and on Thursday and Friday a two-day scientific program will be presented at the Morrison Auditorium in Golden Gate Park.

The guest speakers will include Sir Stewart Duke-Elder, London; A. Franceschetti, Geneva; Giambattista Bietti, Parma; Josef Bock, Graz; Norman Ashton, London; M. Puig Solanes, Mexico City; Hugh L. Ormsby, Toronto; Frederick H. Verhoeff and David G. Cogan, Boston; Alan C. Woods and Jonas S. Friedenwald, Baltimore; Algernon B. Reese, New York; Derrick Vail, Peter C. Kronfeld, and Georgiana D. Theobald, Chicago; Kenneth C. Swan, Portland, Oregon; Col. John H. King, Jr., Washington, D.C.; and S. Rodman Irvine, Beverly Hills, California. On Thursday evening Dr. Ludwig von Sallmann will deliver the Ninth Francis I. Proctor Lecture.

MISCELLANEOUS

AWARDED LESLIE DANA MEDAL

At a meeting of the National Society for the Prevention of Blindness held recently in Saint Louis, Mrs. Audrey Hayden Gradle was awarded the Leslie Dana Medal of the Saint Louis Society for the Prevention of Blindness for outstanding work in the cause of blindness prevention. At the 1946 Chicago meeting of the American Academy of Ophthalmology and Otolaryngology, Mrs. Gradle's husband, the late Dr. Harry S. Gradle, was also presented with the Leslie Dana Medal.

In 1952, at the meeting of the IV Pan-American Congress of Ophthalmology in Mexico City, Mrs. Gradle was elected an honorary member of the Pan-American Association and was made executive director of Prevention of Blindness activities.

Howe Lecture

Dr. Frank B. Walsh, associate professor of ophthalmology, Johns Hopkins University and Hospital, Baltimore, gave the Howe Lecture of Ophthalmology, on April 20th, at the Harvard Medical School. The lecture is given under the auspices of the Howe Laboratory of Ophthalmology. The subject of Dr. Walsh's address was "A clinical study of several third-nerve lesions."

Societies

Nassau speaker

Dr. Isadore Givner, New York, presented a paper on "Ocular infections" before the Nassau Ophthalmological Society on April 26.

New York officers

Officers of the New York Ophthalmological Society for 1954 are: President, Dr. Osborn P. Perkins; vice-president, Dr. Brittain F. Payne; secretary-treasurer, Dr. Harold W. Brown. Members of the Committee on Admissions are: Dr. Guernsey Frey, Dr. Gordon M. Bruce, Dr. Frank C. Keil, Dr. Rudolf Aebli, and Dr. Wendell L. Hughes.

United Kingdom meeting

The annual congress of the Ophthalmological Society of the United Kingdom was held at the University of Durham, Newcastle upon Tyne, on April 22nd, 23rd, and 24th. At the opening session, the Nettleship Medal was awarded to Dr. Norman Ashton. On the program were:

Discussion: "Inflammatory vascular diseases affecting the eye," with Mr. R. C. Davenport, Dr. S. P. Meadows, and Prof. J. B. Duguid acting as openers. Two symposiums were held: "The changes in the incidence of the eye affections of coal miners in the last 25 years" (Openers: Dr. Dorothy Campbell, Mr. W. J. Wellwood Ferguson, and Dr. G. I. Scott); and "Gonioscopy after glaucoma operations" (Openers: Mr. H. E. Hobbs and Mr. J. H. Redwood Smith).

Papers were presented by: Mr. A. J. B. Goldsmith, "Cyclodiathermy"; Mr. George Black, "Observations on the diagnosis of orbital tumors"; Dr. G. V. T. Matthews, "Sun navigation in birds"; Mr. Eugene Wolff, "The blood supply of the 3rd, 4th, and 6th cranial nerves"; Mr. D. P. Greaves, "Experimental penetrating keratoplasty"; Mr. E. C. Glover, "The results of cataract surgery at Moorfields in 1949"; and "Congenital vascular veils in the vitreous," Mr. A. MacRae.

Short demonstrations were given by: Mr. A. Seymour Philps and Dr. P. Hansell, "Keratography"; Mr. A. T. G. Evans, "Essential atrophy of the choroid with ataxia and a strong family history of similar conditions" (introduced by Mr. H. Vernon Ingram); Mr. J. Frankenthal, "Large cyst of the iris following lens extraction" (introduced by Mr. Arthur Smith); Mr. P. J. L. Hunter, "An unusual bilateral case of lymphosarcoma of the orbit"; Mr. E. F. King, "Two cases of secondary carcinoma of the choroid"; and Mr. P. D. Trevor-Roper, "Symmetrical metastases in the angles of the anterior chambers."

The following films were shown: "Retinal detachment," Prof. H. M. Dekking; "Advancement and resection of the levator for correction of congenital ptosis," Dr. G. I. Scott; "Keratoplasty," Mr. A. G. Leigh; "The miner at work," Mr. H. Vernon Ingram.

Pudget Sound academy

Officers of the Puget Sound Academy of Ophthalmology and Otolaryngology are: President, Dr. Carl D. F. Jensen; president-elect, Dr. William H. Ludwig; secretary-treasurer, Dr. Willard F. Goff.

Kansas City meeting

On the ophthalmology program of the April 5th and through April 9th meeting of the Kansas City Society of Ophthalmology and Otolaryngology held at the University of Kansas Medical Center, Kansas City, Kansas, were:

Dr. Larry L. Calkins, "Clinical pathologic conference"; Dr. William F. Hughes, Jr., Chicago, "Differential diagnosis in iritis and uveitis"; Dr. Dick H. Underwood, "Optic-nerve pathology"; Dr. Francis Heed Adler, Philadelphia, "Diagnostic examination in strabismus"; Dr. A. N. Lemoine, Jr., moderator, "Symposium on refraction problems"; Dr. Adler, "Treatment of strabismus"; Dr. James T. Robinson, "Diagnosis and management of ptosis"; Dr. John W. Unruh, "Gonioscopy"; Dr. Hughes, "Treatment of iritis"; Dr. A. N. Lemoine, Jr., moderator, "Symposium on diagnosis and treatment of acute ocular pain"; Dr. Leonard Christensen, "New drugs in the treatment of glaucoma."

Dr. A. N. Lemoine, Jr., "Neurologic fields"; Dr. Otto H. Elser, "Diabetic changes in the eye"; Dr. Christensen, "Pathologic studies of ocular surgery complications"; Dr. Jerome A. Hilger, "Endolymphatic hypertension and its treatment"; Dr. G. O. Proud, moderator, "Case presentation and panel discussion"; Dr. Hilger, "Facial injury."

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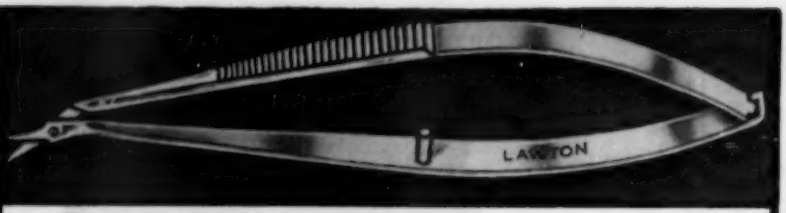

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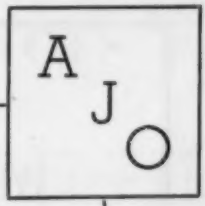

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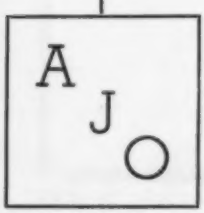

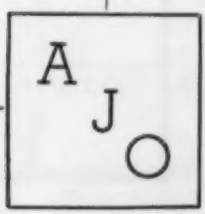

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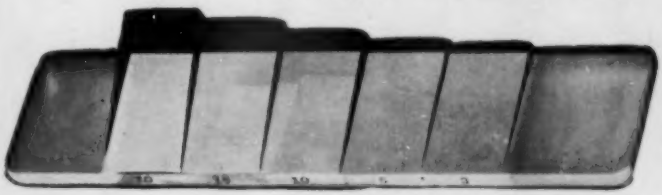

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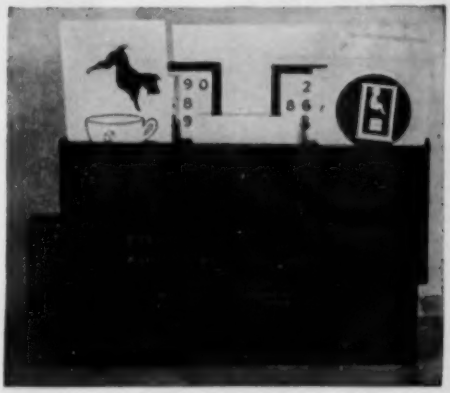

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